VOLUME 31 — 1961-1962

THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY

Published for the Royal Australasian College of Surgeons

CONTENTS

VOLUME 31 — 1961-62

AUGUST, 1961

NOVEMBER, 1961

THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY

FEBRUARY, 1962

MAY, 1962

The Australian and New Zealand
JOURNAL OF SURGERY

Vol. 31 — No. 1 AUGUST, 1961

RETROMANDIBULAR PAROTID TUMOUR*

By T. H. Ackland
Melbourne

PAROTID tumours arising in the retromandibular portion of the gland and presenting primarily in the lateral pharyngeal wall, are sufficiently rare for most experienced surgeons never to have encountered an example. Although their general features and pathological anatomy have now been well established, difficulty and delay in diagnosis are still very likely. Moreover, no agreement has been reached on the important question of the best surgical approach for their removal.

The purpose of this paper is to place on record clinical details of one such case and to give support to the view that a surgical approach from below the mandible is the route of choice. In this instance, a tumour which had remained undiagnosed for a long time and had been considered inoperable by any manoeuvre other than a difficult and hazardous intraoral attack, was removed with surprising ease by a submandibular incision. It seems extremely likely that all of these tumours are best dealt with by this technique.

The first author to mention parotid tumours presenting in the throat was Proby, who described 4 cases in 1924. Stein and Geschickter, in 1934, mentioned that 7 of 113 cases of parotid tumour (5.2 per cent.) appeared "in the throat," but did not describe any special characteristics of this group. A great deal of confusion has arisen from an over-awareness of the fact that mixed salivary tumours may arise in the minor salivary glands of the palate and pharynx, as well as in the parotid, submaxillary and sublingual glands. The result has been that the possible parotid origin of a tumour appearing in the tonsillar region has tended to be overlooked. In fact, not a few writers do not even mention the parotid as a source of such tumours appearing here (e.g. New and Childrey, 1931). The distinction is not only academic, for these tumours, though they are deeply situated, can be removed with comparative ease.

Havens and Butler in 1955 gave an accurate anatomical description of retromandibular parotid tumours but strangely enough reached the surely wrong conclusion that they were of pharyngeal origin. It remained for Patey and Thackray, and Patey and Ranger, in 1957, to give the first clear description of the exact way in which these tumours arrive at the lateral pharyngeal wall.

Surgical Anatomy

The pathological anatomy of retromandibular parotid tumour is displayed in Fig. I and can be readily understood when it is remembered that a small part of the parotid gland curls around the posterior border of the mandible. Although this part extends for a variable distance medially, it is very doubtful whether its deepest limit normally reaches the pharyngeal wall. The portion of the gland which is usually innermost lies in a narrow passage which Patey and Thackray have aptly termed the stylomandibular tunnel (Fig. II); at this point it closes, in a plug-like fashion, the outer entrance to the lateral pharyngeal space. This space is situated lateral to the palate and the upper part of the pharynx and it now seems clear that those parotid tumours which are found here, grow into this region from a more lateral point and do not arise locally. The expansion

*Received for publication 7th July, 1960.

of a parotid tumour medial to the stylo-mandibular tunnel into the lateral pharyngeal space is determined, as Patey and Ranger point out, by the tumour chancing to begin in the very small and deepest piece of the gland which plugs that tunnel. Any more superficial tumour origin will result in growth in an outward direction to produce an ordinary variety of parotid tumour. Patey and Ranger conclude, "The small bulk of this tissue relative to that of the parotid gland as a whole, would explain, on mathematical grounds, the great rarity of pharyngeal extensions of parotid tumours."

visible externally as a subcutaneous mass just in front of the angle of the mandible.

CLINICAL FEATURES

Early in its history, a pharyngeal extension of a parotid tumour will cause no symptoms but it will nevertheless result in inward bulging of the tonsil and soft palate. At this stage it may be discovered accidentally in the course of a routine throat examination. Growth is usually very slow and some years may pass without the patient experiencing much trouble. Later on, as the tumour increases to its usual large size, symptoms such as difficulty in swallowing, a feeling of "something in the throat," or alteration of vocal tone may develop. Gross displacement of the soft palate, tonsil and uvula is then to be seen (Fig. III) and, although ulceration with bleeding appears to be an unusual complication, it happened in the case here reported. Other symptoms which have occurred in some of the documented cases are due to pressure on the mandibular division of the fifth cranial nerve, with resulting neuralgia and blockage of the Eustachian tube with middle ear deafness. However, pain is usually absent, as also is facial nerve involvement.

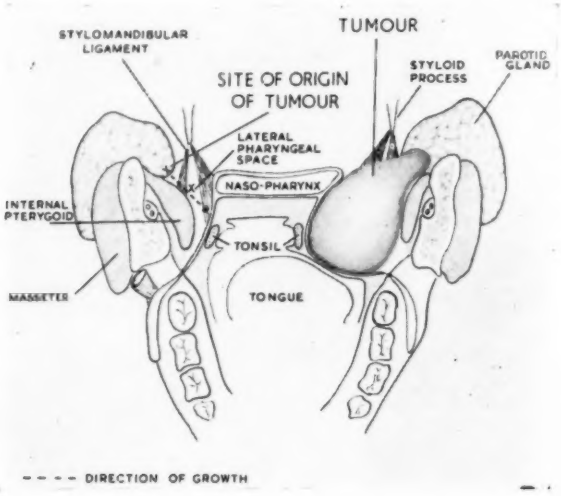


FIG. I. The anatomy of retromandibular parotid tumour.

The term "dumb-bell" tumour has been applied to these neoplasms, since there may be a portion superficial, as well as a portion deep, to the stylo-mandibular tunnel with a constricted isthmus between these two. But this shape will not be assumed if the whole of the growth is deep to the stylo-mandibular tunnel. This was so in the case here described, and it was also so in all of the remarkable series reported by Morfit (1955). There seems little doubt that the larger the tumour, and the longer its history, the less likely it is to have a "dumb-bell" shape, for the stylo-mandibular ligament will gradually stretch, allowing the growth to assume globular or ovoid proportions. Further enlargement may then occur not only medially, but also downwards, so that the tumour may finally become

In most cases, since the ascending ramus of the mandible prevents any lateral expansion of the tumour, there is nothing abnormal

to be seen or felt in the parotid region; but, as has already been indicated, very large examples may grow downwards to become visible and palpable below the angle of the mandible. The mass may be ballottable between internal and external examining fingers, but this sign, which has been stressed by Patey, Thackray and Thompson, is certainly not to be elicited in all cases. Indeed, one would not expect that a tumour situated entirely deep to the mandible, especially one with no external "dumb-bell" protusion, would be ballottable through a narrow sylomandibular tunnel.

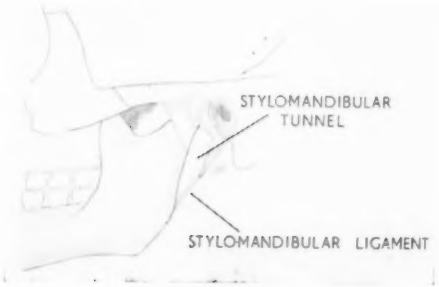


FIG. II. The stylomandibular tunnel.

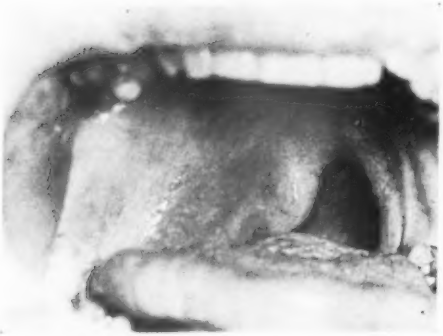


FIG. III. Inward bulging of the tonsil and soft palate.

DIAGNOSIS

If clinical examination establishes that there is an extratonsillar mass in the lateral pharyngeal space, with a history of very slow growth, the diagnosis of retromandibular parotid tumour may be made with considerable confidence.

Although a variety of tumours may develop from parotid tissue, the commonest is "mixed" parotid tumour and any of these neoplasms, innocent or malignant, may occur in that deepest portion of the gland which is here being considered.

On the other hand, a group of tumours which are unrelated to the parotid gland may produce clinical signs identical with those which have been described. These include "latero-pharyngeal" fibroma, fibrolipoma, neurofibroma, lymphoma and neurinoma; but each of these is undoubtedly much less common than pharyngeal parotid tumour. Aneurysm of the external carotid artery and carotid body tumour have also been reported by Morfit as causing inward bulging of the lateral pharyngeal wall, but the presence of expansile pulsation should reveal the true diagnosis in the former case. Tumours of the minor salivary glands can of course occur anywhere in the whole mouth cavity, and are not confined to the region outside the lateral pharyngeal wall.

When a tumour such as lymphosarcoma arises within the tonsil itself, this fact can usually be established by careful inspection, for the appearance is quite different from that produced by the pushing inwards of a normal tonsil, palate and uvula by a mass of extrinsic origin. Finally, the diagnosis of peritonsillar abscess may be suggested by the quinsy-like bulging which is seen but although this mis-diagnosis has been made on some occasions, the absence of pain and fever, together with the long history, should always enable this condition to be excluded.

TREATMENT

Reference has already been made to the differing advice given by those who have written on this subject concerning the most desirable surgical approach. Patey and Thackray, who have dealt with 4 cases, advise total parotidectomy with fracture of the styloid process and preservation of the facial nerve, as a preliminary to reaching the parapharyngeal tumour; but Thompson, who has also operated on 4 cases, advises an oral approach. Morfit, who has had the unique experience of dealing with no less than 12 retromandibular parotid tumours, advocates the submandibular route—a method of attack which is perhaps a little surprising, and not sufficiently obvious to suggest itself at once.

For mixed parotid tumours is general, agreement has been reached that simple enucleation is inferior to parotidectomy, in which

the tumour is removed together with a protecting layer of normal gland around it. However, as Fig. I shows, retromandibular parotid tumours are not surrounded by a capsule of normal gland except on the smallest part of their circumference. It is therefore clear that nine-tenths of the mass must always be enucleated digitally or otherwise, in any case, whatever approach is employed. For this reason, total conservative parotidectomy by a wide external exposure, with fracture of the styloid and division of the stylomandibular ligament, as suggested by Patey and Thackray, seems unnecessarily complicated. The usual appreciable incidence of permanent facial nerve paresis after this operation (9 per cent., McCune) would certainly be increased in dealing with these particular tumours deep to the facial nerve. Moreover, there is no record of a recurrence following local removal of a retromandibular parotid tumour.

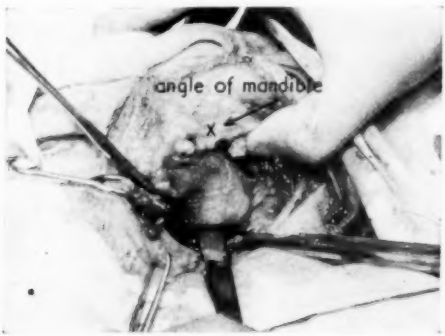


FIG. IV. First view of the lower pole of the tumour.

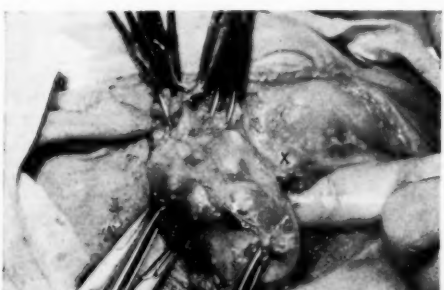


FIG. V. Delivery of the tumour.

A strong argument against the oral approach is provided by the fact that these tumours are usually very large, for example 3 by 2 inches, so that their delivery through the open mouth would be a matter of some difficulty, quite apart from the problems of control of haemorrhage and the maintenance of airway. Nevertheless, a recent paper by Thompson advises a surgical approach through the mouth with enucleation similar to that used in prostatectomy. This seems a very unattractive proposition to the writer when there exists a superior route which gives direct vision at all stages of the operation.

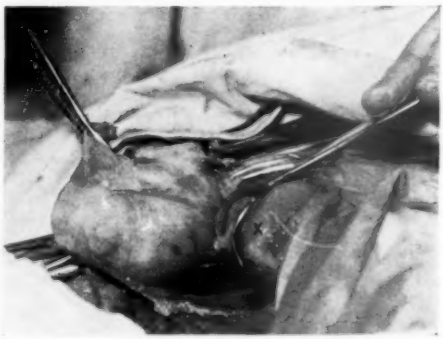


FIG. VI. Delivery of the tumour.

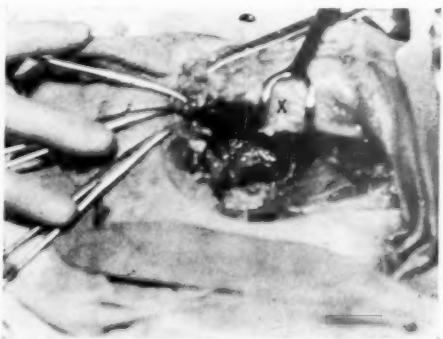


FIG. VII. The cavity remaining.

Morfit's exposure of the pharyngo-maxillary space through the submaxillary triangle is an example of ingenious applied anatomy which provides the easiest and best approach. The incision is the one routinely made for a submaxillary gland dissection, with a little backward extension below the ear. The skin flap is mobilized upwards with preservation of the mandibular branch of the facial nerve.

The submaxillary salivary gland is then removed in order to obtain a wider view, after which the external carotid artery may be ligated to control bleeding in the ensuing steps of the operation. The lower pole of the tumour mass is now clearly in view, deep to and below the angle of the mandible (Fig. IV). At this stage tissue forceps are applied to the capsule of the tumour to assist in its delivery, which is now likely to be

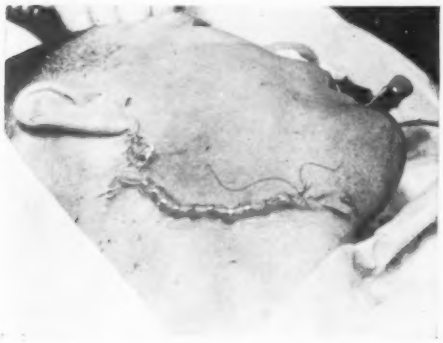


FIG. VIII. The incision sutured with drainage.

accomplished without difficulty (Figs. V and VI). An unusual finding will be a "dumb-bell" tumour with incarceration of the portion superficial to the stylomandibular tunnel. Should this problem be encountered, the styloid process and facial nerve must be exposed and the former divided to allow delivery of the whole mass. A large cavity results, so that drainage is required for several days (Figs. VII and VIII). The tonsil, palate and uvula will be found to have at once assumed a normal appearance (Fig. IX).

CASE HISTORY

M.T.J.C., aged 35 years, was first seen on 2nd December, 1957, when he was referred for an opinion concerning an "enlarged tonsil' which had been present for at least ten years. He complained of no pain, nor dysphagia, but had found that he would get "out of breath" and "unable to breathe properly" when playing tennis or cricket.

Clinical examination showed gross inward bulging of the left tonsil with extreme distortion of the palate, the uvula being pushed into contact with the opposite tonsil. There was no visible enlargement of the parotid region, but there was a vague suspicion of a deep mass on palpation behind the angle of the mandible. A biopsy of this area was attempted, but yielded only parotid gland tissue. Shortly after this, the patient spat up some bright blood and was then admitted to hospital where it was found that shallow ulceration was present behind the tonsil. Small bleedings recurred on several occasions and then ceased. Further biopsy of the tonsillar region was suggested but rejected as being liable to cause further serious haemorrhage. Two courses of deep X-ray therapy were given without a diagnosis being made, but there was no alteration in the size of the tumour. Two months later the tonsillar bulging was seen to be unaltered but the mucosal ulceration had healed. Up to this time, although the patient had been seen by many senior clinicians and had been presented to a clinical meeting, no diagnosis other than "tumour in the tonsillar region" had been made. It was at this stage that Morfit's paper came to the writer's attention and it was realized that this was in fact a typical case of retromandibular parotid tumour. Operation was performed on 7th June, 1958, using the submaxillary approach described above and a tumour the size of a large lemon was delivered with amazing ease, without preliminary external carotid ligation having been performed. Microscopic examination of the specimen showed that it was a mixed parotid tumour. Convalescence was uneventful, except for the fact that full opening of the mouth was not attained for six months and two years later the patient remains free from any evidence of recurrence.

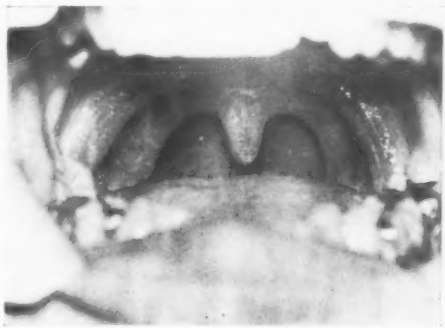


FIG. IX. The normal post-operative intra-oral appearance.

REFERENCES

CRAMMER, L. R. (1958), *Ann. Otol.*, vol. 67, page 178.

HAVENS, F. Z. and BUTLER, L. C. (1955), *Ann. Otol.*, vol. 64, page 457.

MORFIT, H. M. (1955), *Arch. Surg.*, vol. 70, page 906.

MCCUNE, W. S. (1951), *Arch. Surg.*, vol. 62, page 715.

NEW, G. B. and CHILDREY, J. H. (1931), *Arch. Otolaryng.*, vol. 14, page 596.

PATEY, D. H. and RANGER, I. (1957), *Brit. J. Surg.*, vol. 45, page 250.

—— and THACKRAY, A. C. (1957), *Brit. J. Surg.*, vol. 44, page 352.

PROBY, H. (1924), *Arch. int. Laryng.*, vol. 3, page 302.

STEIN, I. and GESCHICKTER, C. F. (1934), *Arch. Surg.*, vol. 28, page 492.

THOMPSON, N. (1960), *Brit. J. Surg.*, vol. 47, page 314.

GASTRO-OESOPHAGEAL CARCINOMA IN HIATAL HERNIA*

By I. McConchie

Royal Melbourne Hospital

IN 1958, Adler and Rodriguez reviewed the English language literature reporting cases suffering from both hiatal hernia and malignancy of the stomach or oesophagus. They found 190 such case reports, of which the biggest personal series were those of Tanner (1955) who described 25 cases, and Dunlop (1956) who reported that 10 of his 141 patients with hiatal hernia had an associated gastro-oesophageal carcinoma. They brought the total of such case reports to 212 by adding 22 cases of their own, approximately 2.5 per cent. of 814 patients with hiatal hernia admitted to their hospitals between 1947 and 1958. Most of the malignancies were adenocarcinomata involving the herniated gastro-oesophageal junction. A smaller number were squamous-celled carcinomas of the lowermost oesophagus, just above the herniated cardia. A very few of these cases had carcinoma of the upper oesophagus or lower stomach.

In my own series of 60 patients, whose symptoms associated with their sliding hiatal herniae warranted operation, are 5 patients suffering from an associated carcinoma in the herniated pouch of stomach or in the oesophagus immediately above the herniated cardia. In other words, in my experience, if a patient with an hiatal hernia has symptoms sufficiently severe to warrant operation, there is an 8 per cent. chance that this patient has an associated gastro-oesophageal carcinoma.

It seems likely that the association of malignancy high in the gullet or low in the stomach, with hiatal hernia, is merely a coincidence. However, an area of peptic oesophagitis, well above a herniated cardia, is fairly common and such an oesophageal abnormality may predispose to the development of a high carcinoma of the oesophagus. I have not seen such a case.

There has been much discussion about whether the association between gastro-oesophageal cancer and hiatal hernia is co-incidental, whether the hernia predisposes to the development of the carcinoma or whether the carcinoma causes herniation of the cardia. Solution of this problem seems less important than awareness of the fact that association of the two lesions is fairly common. This is one of the several reasons why all patients with a symptomatic hiatal hernia should have a diagnostic oesophagoscopy and biopsy.

It is also important to realize that an hiatal hernia, detected by X-ray, may not be causing symptoms. An associated carcinoma, not clearly shown in X-rays, may be responsible for the dyspepsia and dysphagia. This is yet another reason why all patients with symptomatic hiatal hernia, should have an oesophagoscopy and biopsy.

An associated carcinoma of the cardia with submucosal involvement of the lower gullet may not be diagnosed even by oesophagoscopy and biopsy. This is one of many reasons why, if symptoms persist in spite of medical treatment, exploratory operation should be performed.

Most of my patients had a long history of hiatal hernia dyspepsia. They then developed new dyspeptic symptoms, or more troublesome dysphagia, due to the growing carcinoma. Investigation of the cause of these new symptoms was delayed for a long time because they were thought, by the patient or his doctor, to be due to a long-standing hiatal hernia. At operation all of these patients had very extensive gastro-oesophageal carcinoma, hence their depressingly short survival time, after operation.

Case Reports

Case 1

A.R., male, 61 years, for many years had complained of epigastric fullness and belching immediately after meals. He had frequently regurgitated after meals.

*Received for publication, 27th July, 1960.

For six months solid food had occasionally stuck at the level of the xiphisternum. It then passed slowly down into the stomach. During the two weeks prior to admission to hospital he had not been troubled by any dysphagia.

A barium swallow and meal on 27th December, 1956, showed a three-quarter inch long stricture of the lower gullet immediately above a small sliding hiatal hernia. There was a filling defect in the herniated pouch of stomach (Fig. 1). He was thought to have either an hiatal hernia with a peptic stricture of the gullet or a gastro-oesophageal carcinoma associated with an hiatal hernia.

FIG. 1. Hiatal hernia with stricture of lower gullet.

An oesophagoscopy was done on 10th January, 1957. The cardia was seen, above the diaphragm, 38 cms. below the upper gum margin. Changes typical of superficial peptic oesophagitis were seen in the lower gullet. The lower gullet and cardia were easily dilated up to size 28 English Hurst's mercury-loaded bougie. The whole appearance did not suggest malignancy. A biopsy was taken from the cardia and it did not show evidence of malignancy.

It was therefore thought that he probably had a hiatal hernia with a peptic stricture of the lower gullet. As I regard a complicating stricture as an indication for operation in patients with hiatal hernia, I advised operative repair of the hiatal hernia.

Operation was performed on 18th January. The left chest was entered through a lower left thoracotomy incision. The cardia was herniated 2" into the mediastinum. A malignant mass was present in the herniated stomach and lower oesophagus. It extended down into the upper stomach below the diaphragm. Malignant glands were present in the lower mediastinum. The diaphragm was opened in front of the oesophageal hiatus and the upper abdomen explored. Malignant glands were present around the coeliac axis and the hepatic artery and the growth was regarded as inoperable. A gland removed from around the coeliac axis was invaded by adenocarcinoma. As he could swallow reasonably comfortably no palliative by-passing anastomosis was done.

After operation he dilated his malignant stricture by swallowing a Hurst's bougie daily. This enabled him to take a soft diet reasonably comfortably for a few months. However, by 1st August, 1957, he could not pass the bougie through the stricture and, by 30th August, he could not swallow liquids.

At this time a barium swallow showed complete oesophageal obstruction. Oesophagoscopy showed an obviously malignant stricture which could not be dilated.

A laparotomy was therefore done on 30th August, when multiple metastases were found in the liver. A small, high gastrotomy opening was made and a small Hurst's bougie was passed upwards, through the malignant stricture, into his mouth. Two heavy sutures attached the end of a flanged Portex tube to the Hurst's bougie. The bougie and the attached Portex tube were then pulled down through the stricture until the collar of this tube rested immediately above the growth. The sutures were picked up in the stomach and they were then sewn through the full thickness of the stomach wall to prevent vomiting of the Portex tube. The patient swallowed a soft diet, without any difficulty, until he died of liver metastases in December, 1957.

Comments on Case 1

This patient's familiarity with his long-standing hiatal hernia dyspepsia made him contemptuous of his increasing dysphagia. The dysphagia had been present for six months before he complained and was investigated. Consequently his carcinoma had advanced to the stage of inoperability by the time operation was performed.

Oesophagoscopy and biopsy failed to confirm the presence of a carcinoma. When the malignancy commences in the upper stomach and involvement of the cardia and lower gullet is confined to the submucosa, the growth will rarely be seen at oesophagoscopy and the biopsy specimen will often be too shallow to reveal the growth. Even if this investigation fails to prove the presence of an associated carcinoma, exploratory operation should be performed if X-ray suggests the possibility of carcinoma, a stricture is

present in the lower gullet, or if symptoms persist in spite of medical treatment. If these indications for operation are followed, few gastro-oesophageal carcinomata, associated with hiatal herniae, will be overlooked. The indications for operation in this man were the presence of a stricture and an X-ray suggestive of carcinoma.

A stricture of the oesophagus, malignant or benign, that cannot be dilated through an oesophagoscope, can often be dilated and intubated by the retrograde method used in this patient.

Although this patient was found to have an inoperable carcinoma, he lived for eleven months after his exploratory operation and the institution of palliative treatment. Thus he survived longer than the other 4 members of this group, who had their malignancy resected.

Case 2.

H.W., male, 60 years, for years had suffered from heartburn at night and an hiatal hernia had been diagnosed on X-ray two years previously.

For six months the passage of food through the lower half of his gullet caused pain in the retrosternal region. The food often stuck at the level of his xiphisternum causing retrosternal and interscapular pain. When he managed to belch up some wind, the food passed down and the pain disappeared. Meal times had become so distressing that he rarely started and never finished a meal. He had lost 21 pounds in weight in the previous six months.

X-rays showed a small hiatal hernia, above which was an irregular stricture of the lower gullet. A cholecystogram showed a non-filling gall-bladder with calculi.

At oesophagoscopy on 21st July, 1957, what looked like superficial peptic oesophagitis was seen. The lower oesophageal stricture was easily dilated and a biopsy of the lower gullet showed no evidence of carcinoma.

He was thought to have an hiatal hernia with a peptic stricture of the gullet and cholelithiasis. It was decided to repair the hiatal hernia and remove the gall-bladder.

On 30th July, Mr. D. Leslie and I operated through a long left thoraco-abdominal incision. The gall-bladder was removed and the common bile duct, which contained calculi, was cleared and drained. A small sliding hiatal hernia was present and the lower inch of the gullet was rigid and densely embedded in the mediastinum. His gullet was mobilized and it was again easily dilated by a bougie passed by the anaesthetist. The stomach felt normal. The lower gullet was thought to be the site of a peptic stricture. An Allison type repair of the hiatal hernia was done after learning that a frozen section of some small hard lower mediastinal glands showed no evidence of malignancy.

Two days after operation, examination of the paraffin section of the removed mediastinal glands showed invasion by squamous celled carcinoma. Therefore, on 12th August, 1957, a partial oesophago-gastrectomy with oesophago-gastric anastomosis was done. A small malignant ulcer was present in the strictured part of the lower gullet, not seen at oesophagoscopy because the oesophagoscope could not be passed into the stricture.

The patient died suddenly of a massive pulmonary embolus on the twelfth post-operative day.

Comments on Case 2

This patient also failed to seek investigation of his dysphagia for six months, thinking it was just another symptom of the hiatal hernia that had been diagnosed by X-ray previously. By the time operation was performed he already had the malignant mediastinal gland invasion which is usually followed by mediastinal recurrence of carcinoma after oesophagectomy.

The difficulty of diagnosing gastro-oesophageal carcinoma in a patient with a hiatal hernia is shown in this case. The condition was not diagnosed even after oesophagoscopy with biopsy and thoracotomy with frozen section of the involved mediastinal glands. The patient was thought to be suffering from, and was treated for, a peptic stricture of the gullet above a hiatal hernia until the result of paraffin section of his mediastinal glands was available.

Case 3

G.C.B.M., male, 50 years, for as long as he could remember had found it necessary to eat small meals very slowly, otherwise he felt distended and food stuck at the level of his xiphisternum.

For about six months he had suffered pain as food passed through the lower gullet. For one month he had no appetite at all. For two weeks he had suffered constant epigastric pain. He had lost a lot of weight.

He looked ill, had a persistent temperature of 102° F, and had gross finger clubbing of recent origin. No other abnormality was found on physical examination.

X-ray of his chest showed no abnormality, in particular no metastases were seen to account for his finger clubbing. A barium meal showed a large sliding hiatal hernia with a filling defect in the herniated stomach and a soft tissue mass in his mediastinum around the herniated stomach (Figs. IIa and IIb).

Oesophagoscopy revealed a fungating carcinoma in the lower gullet which, on section, proved to be an anaplastic carcinoma of the stomach extending up into the oesophagus.

Operation by Mr. E. S. R. Hughes and myself on 2nd August, 1956, revealed a very extensive carcinoma involving the lower gullet and herniated stomach, upper abdominal stomach, mediastinal and coeliac axis glands. The lower gullet and upper stomach, along with the spleen and distal half of his pancreas were removed and an oesophago-gastrostomy was performed. No metastases were found in the liver or left lung, so the finger clubbing remained unexplained. After operation his fever disappeared but finger clubbing persisted.

FIG. IIa. Barium meal, antero-posterior view, showing large hiatal hernia and surrounding soft tissue mass in the mediastinum.

On the fifteenth post-operative day he developed an anastomotic leak with an oesophago-cutaneous fistula. This closed in three weeks and he subsequently had no dysphagia.

Three months after operation a large, hard, knobbly liver was detected and needle biopsy confirmed the presence of metastases in the liver. He died of metastatic carcinoma four months after operation.

Comments on Case 3

It is not surprising that this patient did not seek investigation and treatment of his dyspepsia and dysphagia until his gastro-oesophageal carcinoma was very extensive, because symptoms due to his growth differed very little from the long-standing symptoms due to his hiatal hernia.

An unusual X-ray picture in this case was the presence of a soft tissue shadow in the lower mediastinum due to extramural extension of the carcinoma of the herniated stomach.

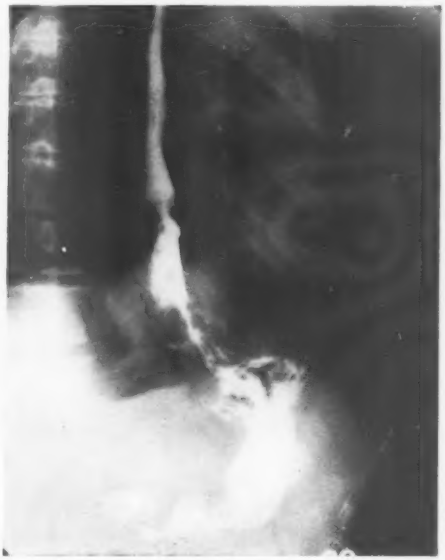


FIG. IIb. Barium meal, lateral view, showing large hiatal hernia with filling defect in the herniated stomach and soft tissue mass in the lower mediastinum.

I presume the gross finger clubbing, obvious when he was first seen, was due to microscopic liver metastases. These were not detected at operation but a large malignant liver was obvious just three months after removal of the primary growth.

Cases 4 and 5

G.N., male, 64 years, and V.M., male, 77 years, both had similar histories, X-ray findings, oesophagoscopy and biopsy findings operative findings and operative procedures.

Neither had any dysphagia or dyspepsia until one month before I first saw them. They both complained of painful blockage of their lower gullet by food. The food passed on slowly and their pain then passed off.

X-rays of both patients showed a small sliding hiatal hernia with a stricture of the lower gullet immediately above the cardia (Figs. III and IV). In both cases the stricture was at least partly due to spasm because it distended during different phases of swallowing.

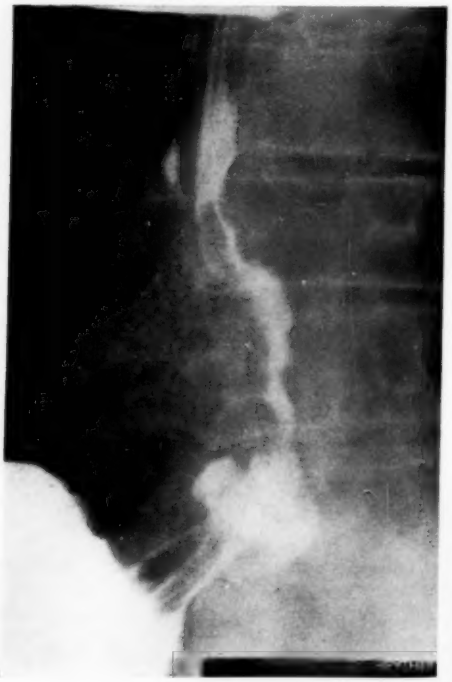


FIG. III. Barium meal showing hiatal hernia and stricture of the lower gullet.

In neither case was any abnormality seen at oesophagoscopy, except that the cardia was above the diaphragm, 37 cms. below the upper gum margin. Large Hurst's bougies passed easily through the cardia. In each case a biopsy from the cardia was taken and this showed adenocarcinoma in the deeper layers of the biopsy specimen.

At operation in each case, a carcinoma of the lower gullet and upper stomach, with involvement of the mediastinal and coeliac axis glands was found. In each case a radical resection of the involved gullet, stomach, glands, spleen and distal pancreas was performed and, in each case an oesophago-gastrostomy was done. The operation on G.N. was performed by Mr. E. S. R. Hughes and myself, and Mr. G. McKenzie assisted me with the operation on V.M.

V.M. died on 2nd June, 1959, of liver metastases, seven months after operation. G.N. is at present dying, fourteen months after operation, of recurrent carcinoma in the distal stomach, demonstrated at a recent oesophagoscopy (Fig. V).

Comments on Cases 4 and 5

Neither of these patients had any symptoms due to their hiatal hernia. Both sought treatment within a month of the onset of their dysphagia. Yet, in both cases, very extensive carcinoma was found at operation.

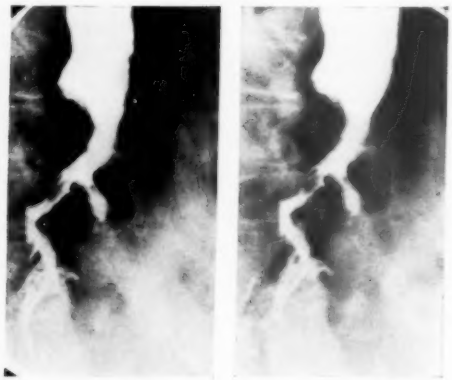


FIG. IV. Barium meal showing small hiatal hernia and stricture of the lower gullet.

FIG. V. Barium meal showing filling defect due to recurrent carcinoma in a stomach remnant.

In both of these cases the value of biopsy at the time of oesophagoscopy is demonstrated. In neither of them did the X-ray or oesophagoscopy findings suggest anything

more sinister than hiatal hernia with a spastic stricture of the lower gullet. However, biopsy of the cardia, in each case, showed an adenocarcinoma of the stomach involving the deeper layer of the lower gullet.

SUMMARY

Gastro-oesophageal carcinoma is a lesion fairly commonly associated with sliding hiatal hernia. Eight per cent. of my patients with hiatal hernia, whose symptoms were sufficiently severe to warrant operation, had the double lesions.

The possible association of the two lesions is one of several reasons why all patients with hiatal hernia, who suffer from dyspeptic symptoms, dysphagia or blood loss, should have oesophagoscopy with biopsy.

In the presence of hiatal hernia it may be difficult to diagnose an associated gastro-oesophageal carcinoma. Symptoms and X-ray and oesophagoscopy appearances due to associated carcinoma, can readily be attributed to the hiatal hernia and peptic oesophagitis. For this reason exploratory operation should be done if symptoms, thought to be due to hiatal hernia, do not completely disappear with medical treatment.

Three out of five of the patients in my series had long-standing dyspepsia due to their hiatal hernia. They did not seek investigation of their new symptom, dysphagia, for several months because they wrongly thought it was due to their long-standing hiatal hernia, whereas it was due to the associated carcinoma. Consequently, at operation a very extensive carcinoma was found. This delay in diagnosis of the malignancy is partly responsible for the short post-operative survival time.

REFERENCES

ADLER, R. H. and RODRIGUEZ, J. (1958), *J. thorac. Surg.*, vol. 37, page 553.

DUNLOP, E. E. (1955), *Gastroenterologia*, vol. 86, page 287.

TANNER, N. L. (1955), *Lancet*, vol. 2, page 1050.

PRIMARY CHORIONEPITHELIOMA OCCURRING IN AN INTERSTITIAL PREGNANCY*

By Robert F. Zacharin

Royal Melbourne Hospital

PRIMARY tubal chorionepithelioma and interstitial pregnancy are each rare conditions and a survey of available world literature suggests their combination is unique.

Case History

Mrs. I.K., aged 43 years, first attended the Royal Melbourne Hospital on 18th May, 1959, complaining of severe pain in the right iliac fossa for three days.

She was a confirmed chronic alcoholic, and had been separated from her husband for five years. She had two children, aged 15 and 20 years. There had been no other pregnancies.

Menstruation had been normal until six months ago, when a very heavy menstrual loss occurred. This had persisted for several weeks, and was followed by complete amenorrhoea. Four months ago, curettage by her local doctor revealed no apparent abnormality.

Due to the patient's sluggish memory, the history was obtained with difficulty. For one month she had been aware of a lump in the lower abdomen, and for the past three days had noticed a constant, localized and severe pain in the right iliac fossa. She denied the possibility of pregnancy, whilst admitting to intercourse on two occasions, nine months and three months ago.

The patient looked pale and ill. An irregular mass arising from the pelvis was outlined in the lower abdomen. Release tenderness was present in this area. Vaginal examination showed the mass was apparently a bulky, irregular, firm uterus, enlarged to the size of an eight or ten week gestation, with a separate but conjoined and tender mass in the right lateral fornix. She experienced momentary faintness following the vaginal examination but recovered quickly.

The diagnosis was considered to be a complicated ovarian cyst, or a fibromyoma, in which degenerative change had occurred. The previous curettage was assumed to exclude intra-uterine pathology.

Operation

She was admitted to hospital and laparotomy was performed on 20th May, 1959. With the abdomen opened through a left lower paramedian incision, a moderate amount of free blood and blood clot was evident. Omentum adhered to a partially ruptured and bleeding, bluish coloured mass distending the right cornual area of the uterus. The uterine body, the left adnexae and the right adnexae distal to the mass, were normal. Both omentum and pelvic peritoneum were normal. At this stage a diagnosis of ruptured interstitial pregnancy was made and total hysterectomy and right salpingo-oophorectomy performed. Convalescence was uneventful and she was discharged on 31st May, 1959.

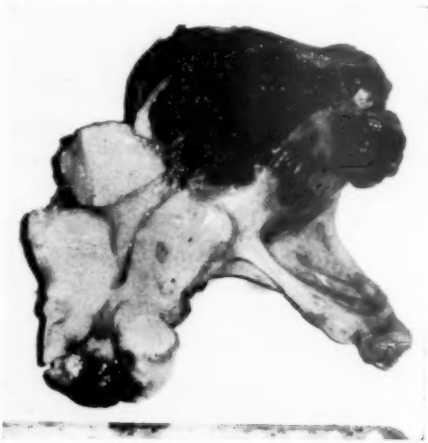


FIG. I. The excised specimen. The normal uterine body, the interstitial pregnancy which is utero-tubal in type and the normal-looking right Fallopian tube emerging from the mass are shown. Several sites of rupture may be seen, together with the thin enclosing wall.

Pathology

Dissection of the excised specimen showed a mass distending and destroying the interstitial portion of the right tube (Fig. I).

The pathology report was as follows:

"Macroscopic examination: The specimen was 15 cm. in length; from the fundus of the uterus projected a large round tumour mass, apparently in a subserosal position, 7.5 cm. in diameter. Most of the tumour was red and friable, but around the edges there were several pale greyish-white nodules. On the posterior surface there was a small subserosal nodule of greyish-brown colour, one cm. in diameter. The right ovary and Fallopian tube were included. The ovary was 1.5 cm. in diameter and contained a soft yellow nodule 0.5 cm. in diameter.

*Received for publication 11th July, 1960.

Microscopic examination: Sections taken from different portions of the mass, show large areas of haemorrhage and necrotic material. Some of this necrotic tissue is obviously syncytial but parts appear to be necrotic chorionic villi. At the edges there are sheets of Langhan's cells, with numerous strands of syncytium, which extend into the vascular spaces of the uterine wall. The syncytium is grossly irregular and fairly extensive, forming fairly thick masses on the surface of the necrotic material.

Chorionepithelioma of the uterus" (Figs. II, III, IV).

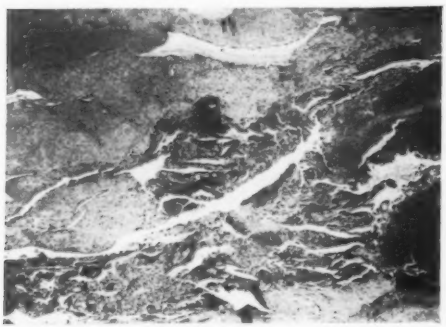


FIG. II. Low-power view of tumour edge in the uterine wall. The syncytial sheets may be seen centrally with blood clot to the right and uterine wall above. (x 25)

Subsequent course

Because of the pathological diagnosis the chest was X-rayed on 27th May. The report stated: "There is a small streaky density at the right apex probably due to an old Koch's lesion, otherwise the lung fields are clear. Post-operative air collection is noted beneath the diaphragm."

The haemoglobin level at this time was 72 per cent. Male toad test on 21st May was positive to a dilution of 1 in 20. With no sign of metastases,

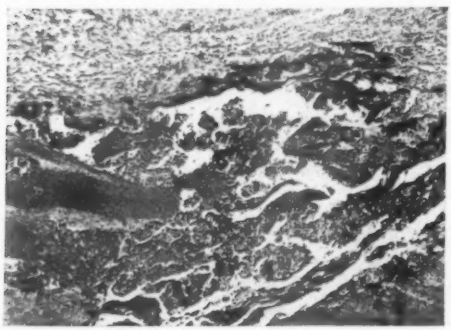


FIG. III. Magnification (x 63) clearly showing both Langhans cells and syncytium invading uterine wall.

it was decided not to institute any specific therapy for the present but in the event of dissemination a course of Methotrexate would be given.

On 18th June the patient was well and the toad test negative in the undiluted specimen. One week later there was a complaint of vague, lower abdominal pain radiating to the epigastrium, and slight tenderness beneath the right costal margin was noted. On 2nd July the chest X-ray remained clear and the toad test was now positive in the undiluted specimen. On 15th July she was readmitted to hospital looking extremely ill. The pain beneath the right costal margin had now been present for three weeks and she complained of abdominal swelling and severe generalized abdominal pain present for three days. Blood pressure was 130/60 mm. of mercury and the pulse rate 128 per minute. The abdomen was grossly distended and particularly tender beneath the right costal margin.

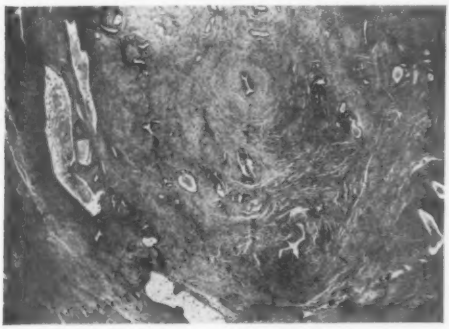


FIG. IV. This interesting section has been taken just immediately distal to the tumour. The normal tubal lumen may be seen centrally and eccentrically about it the characteristic picture of salpingitis isthmica nodosa. This evidence of old chronic salpingitis perhaps explains the origin of the tubal pregnancy.

Operation

Laparotomy showed several litres of blood and blood clot, together with a large profusely bleeding irregular rent on the lower surface of the liver and a large, soft, friable mass was noted in the omentum. Death occurred twelve hours after laparotomy. Toad test was still positive on a specimen of urine taken on admission.

Autopsy

The post-mortem findings were as follows: "The body is that of a middle aged very pale women. There is a recent operation wound in the lower abdomen with sutures still in position and another suprapubic scar. Height 152 cm. Weight 50 kg.

The important findings were as follows: Lungs — Both have a pale mottled red appearance at the rims, but quite livid elsewhere. Here and there, close to the surface there are small haemorrhagic tumours about 1-2 mm. in diameter. The rest of the lung is grossly congested. Heart — The organ is very pale, the muscle flabby. Alimentary canal — There are some adhesions around the pelvis. In the omentum, a large haemorrhagic nodular mass about

5 cm. in diameter, is adherent to the sigmoid colon. Another similar mass, about 2 cm. in diameter, is seen near the splenic flexure. The peritoneum shows a blackish-grey discolouration (Fig. V). Liver — There are three large masses in the liver, two of which are very close to the surface, and one has ruptured. This latter has pushed the right kidney downwards and medially. The ruptured mass is about 7 cm. across, very haemorrhagic and soft, while the other masses have a central fleshy appearance and show less haemorrhage. The remainder of the organ is very pale (Fig. VI). Brain — It is quite soft but shows no evidence of tumour."

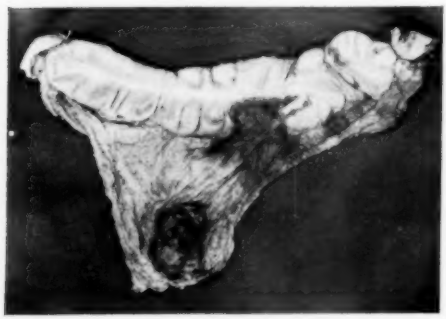


FIG. V. Omental metastases of chorionepithelioma.

Microscopical report: There are several metastases in the lung consisting of sheets of polygonal or spindle-shaped large cells, with a vesicular or compact nucleus, sometimes showing nucleoli. There are a number of mitiotic figures. The cytoplasm is finely granular and acidophil. In some instances tumour cells surround vascular, blod-filled spaces. There is also considerable haemorrhage. Many alveoli are filled with macrophages containing iron pigment (Fig. VII). Similar cells are present in the section taken from the liver. There are no metastases seen in kidney, heart muscle, thyroid, thymus or pituitary.

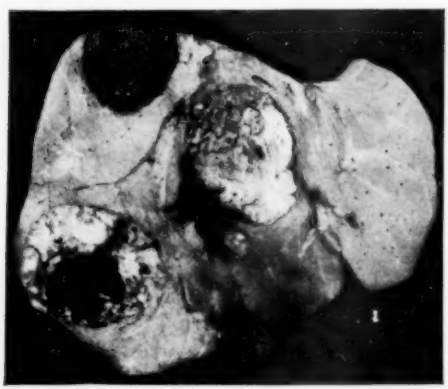


FIG. VI. Hepatic metastases of chorionepithelioma. Rupture has occurred in the largest deposit.

Diagnosis

Interstitial pregnancy, chorionepithelioma, metastases in omentum, liver and lung, with abdominal haemorrhage.

DISCUSSION

Incidence of tubal chorionepithelioma

Sanger (1889) first described chorionepithelioma and Marchand (1895) noted a primary occurrence in the Fallopian tube. Since Marchand's case, less than 60 similar cases have been recorded. Nurnberger (1932) was

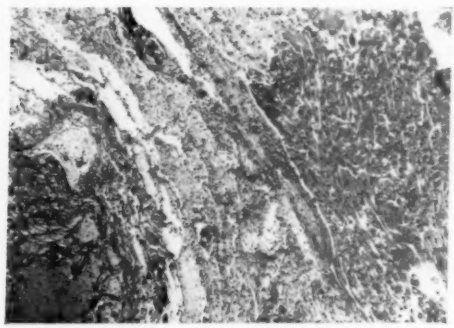


FIG. VII. Pulmonary metastasis. (x 63)

able to collect 33 cases, Williams (1938) 42, Austin (1950) 44 and Heiss (1954) added the fifty-sixth to the literature.

In many large series of published reports of chorionepithelioma there is no case occurring primarily in the tube. Acosta-Sison (1949) reported 70 cases without a tubal primary. Hou and Pang (1956) discussing 28 cases, found no evidence of tubal involvement without a primary uterine tumour. Other notable series reported very few. Park and Lees (1950) 21 in 483 cases, and Brews (1935, 1939) none in 16, then 1 in 24 cases.

The stated incidence varies widely and depends on the criteria for diagnosis, which in earlier papers were less rigid. Hartz (1916) stated 3.5 per cent. of chorionepithelioma occurred primarily in the tube; Heiss (1954) found 1 of 540 tubal pregnancies resulted in chorionepithelioma, and Schaumann and Vogelin (1937) noted the incidence to be 1 in 13,850 pregnancies or 0.007 per cent. Tran Dinh De of Saigon University Hospital, South Vietnam (1959), reported chorionepithelioma was not uncommon in his hospital practice, yet in a public service confining 16,000 females each year,

he has seen only 2 cases (unreported) of primary tubal chorionepithelioma in twelve years practice comprising 150,000 pregnant females.

Incidence of interstitial pregnancy

This is the least common site of tubal pregnancy. The first case reported was that of Schmitt in 1801 (Hyams, 1953), yet by 1932 only 200 cases standing critical analysis had been recorded (Ash, 1932). McIntyre (1922) in a review of tubal pregnancy, noted the very low incidence in the series of Worth, none in 120, Martin, one in 77, and Munro Kerr, one in 80. Wynne (1929) stated an incidence of 10 or 1.65 per cent. in 2,405 tubal pregnancies but figures vary from the Johns Hopkins Hospital reports (1919) of 0.65 per cent. to the 3 per cent. incidence reported by Rosenthal (1896). Ballantyne *et alii* (1940) found the incidence varied from 0.96 to 2 per cent. of all extrauterine pregnancies.

Aetiology of primary chorionepithelioma

Tubal pregnancy would seem to be the most likely cause of this condition, both from a consideration of the symptom sequence and the fact that hydatidiform mole can occur in the tube. Meyer (1919) was able to find 48 such cases and one is mentioned by Blaikley (1935). Pettit (1941) thought it seemed probable from an extensive review of the literature, that hydatidiform mole is present more often in ectopic gestation than is believed, in fact there are few reports of hydatid mole following tubal pregnancy, whereas theoretically it should be more common than chorionepithelioma. Madden (1950) noted there were approximately three times as many reported cases of tubal chorionepithelioma than of tubal hydatidiform mole. It was further stated that pathological examination of all excised tubal pregnancies would probably reveal a high incidence of hydatidiform degeneration.

The transport of villi from a preceding uterine pregnancy, without initial malignant tumour formation in the uterus, is another possible cause of this condition. Fleurent *et alii* (1933) state no such case has occurred with any certainty, yet the case of Williams (1938) with an apparently primary broad ligament chorionepithelioma following a recent abortion in which there was no evidence of tumour formation in the uterus, suggests this mechanism may operate.

Metastatic tubal involvement from a uterine tumour must be excluded before the diagnosis of primary tumour can be considered. Teratogenous origin is usually applicable only to the ovary and finally histogenesis from rudimentary rests or metaplasia has been suggested (Hamdi, 1935).

Diagnosis

Difficulties in diagnosis are stressed by all writers on this subject and indeed no case has been diagnosed correctly before operation. This was stated by Stein (1932) in his review and has remained true. Notwithstanding this fact, there is a definite symptom sequence common to all the reported cases and noted particularly by Fleurent *et alii* (1933), Williams (1938), Pearse and Fraser (1938), Smith and Wertheissen (1941), Madden (1950) and Conill-Serra and Marquez (1957). The symptom sequence is characteristic and covers the three phases of the disease:

1. the signs and symptoms of tubal pregnancy;
2. the period of delay or latent interval *(la periode de latence)*;
3. the phase of tumour growth and dissemination *(la periode tumorale)*.

The patient in this report demonstrated this symptom sequence, in that the initial bleeding followed by the amenorrhoea, almost certainly represented the first two phases. The sudden onset of tumour growth and its dissemination resulting in the fatal issue completed the sequence. Thus, as in all other cases correct diagnosis was theoretically possible.

Mortality

Published mortality figures vary widely but in general the more recently reported cases indicate an almost uniformly fatal outcome. The case of Heiss is an exception. It is likely that the confusion of several pathological entities in earlier papers may account for survival. It may be pointed out that survival without symptoms does not mean recovery, a short or long period of apparent cure before death has been common to most reported cases.

There is no reliable prognostic guide. The Aschheim-Zondek test is used in this regard, but the oft-repeated sequence of a negative test preceding a fatal outcome has been frequently noticed (Williams, 1938; Austin, 1950). It occurred in this case. A negative test frequently follows primary surgery and becomes positive later with metastasis, but a negative test with advancing tumour dissemination is probably due to a highly anaplastic tumour failing to produce chorionic gonadotrophin.

Treatment

Surgical treatment whether limited to partial salpingectomy, or extended to total hysterectomy and bilateral salpingo-oophorectomy, does not seem to appreciably delay the outcome.

Recently, folic acid antagonists have been used in the treatment of disseminated chorionepithelioma. The rationale for this is based on experimental evidence of the vital role played by folic acid in both animal and human foetal tissue growth. Hertz *et alii* (1958) report on 27 cases of disseminated disease treated with varying dosage of 4-amino-N^{10} methyl pteroylglutamic acid (Methotrexate). Twenty-six patients showed remission and in 5 there has been no evidence of recurrence for periods of 8 to 29 months. Both follow-up and response to treatment are assessed on clinical, radiological and biological evidence. Dangerous and occasionally fatal drug reactions and the development of drug resistance make treatment difficult.

SUMMARY

1. A case possibly unique, of chorionepithelioma occurring in an interstitial pregnancy is presented.
2. The case was not diagnosed before pathological examination of the excised material.
3. The case demonstrated the characteristic symptom sequence and fatal outcome.
4. A negative toad test occurred just before the final issue as noted in many previous cases.

REFERENCES

ACOSTA-SISON, H. (1949), *Amer. J. Obstet. Gynec.*, vol. 58, page 125.

ASH, J. E. (1932), *Surg. Gynec. Obstet.*, vol. 54, page 930.

AUSTIN, O. G. (1950), *Ohio St. med. J.*, vol. 46, page 787.

BALLANTYNE, E., MURRAY, K. and FRASER, R. (1940), *Canad. Med. J.*, vol. 43, page 560.

BLAIKLEY, J. B. (1935), *Proc. roy. Soc. Med.*, vol. 28, Part (ii), page 1227.

BREWS, A. (1935), *Proc. roy. Soc. Med.*, vol. 28, Part (ii), page 1224.

—— (1939), *J. Obstet. Gynaec. Brit. Emp.*, vol. 46, page 813.

CONILL-SERA, V. and MARQUEZ, R. S. (1957), *Acta. Ginec. (Madr.)*, vol. 7, No. 6, page 97.

FLEURENT, H., KELLER, R., MEYER, P. (1933), *Bull. Soc. Obstét., Gynéc. Paris*, vol. 22, page 452.

HAMDI, H. (1935), *Ann. Anat. path méd-chir.*, vol. 12, page 493.

HARTZ, H. (1916), *Surg. Gynec. Obstet.*, vol. 23, page 602.

HEISS, H. (1954), *Zbl. Gynäk.*, vol. 76, page 870.

HERTZ, R., BERGENSTAL, D. M., LIPSETT, M. B., PRICE, E. B. and HILBISH, T. F. (1958), *J. Amer. med. Ass.*, vol. 168, page 845.

HOU, P. C. and PANG, S. C. (1956), *J. Path. Bact.*, vol. 72, page 95.

HYAMS, M. N. (1953), *Amer. J. Obstet. Gynec.*, vol. 65, page 697.

MADDEN, S. (1950), *J. Obstet. Gynaec. Brit. Emp.*, vol. 57, page 68.

MARCHAND, F. (1895), *Mschr. Geburtsh. Gynäk.*, vol. 1, page 419.

MEYER, A. W. (1919), *Surg. Gynec. Obstet.*, vol. 28, page 293.

MCINTYRE, D. (1922), *J. Obstet. Gynaec. Brit. Emp.*, vol. 29, page 314.

NURNBERGER, L. (1932), in Stoeckel, W., "Handbuch der Gynakologie." 3te Aufl., 21v., Munchen, vol. 7, page 888.

PARK, W. W. and LEES, J. C. (1950), *Arch. Path.*, vol. 49, page 73.

PEARSE, R. H. and FRASER, C. K. (1938), *Amer. J. Obstet. Gynec.*, vol. 35, page 1046.

PETTIT, M. de W., (1941), *Amer. J. Obstet. Gynec.*, vol. 42, page 1057.

PHILLIPS, H. M. (1911), *J. Obstet. Gynaec. Brit. Emp.*, vol. 19, page 502.

ROSENTHAL, J. (1896), *Gazeta Lakarska, Warszawa.* 2nd ser., pages 16, 1109, 1134.

SANGER, quoted by Teacher, J. H. (1903), *J. Obstet. Gynaec. Brit. Emp.*, vol. 4, page 1.

SCHAUMANN, E. A. and VOGELIN, A. W. (1937), *Amer. J. Obstet. Gynec.*, vol. 33, page 473.

SMITH, T. J. and WERTHEISSEN, N. T. (1941), *Amer. J. Obstet. Gynec.*, vol. 41, page 153.

STEIN, H. E. (1932), *Amer. J. Obstet. Gynec.*, vol. 23, page 416.

TRAN DINH DE (1959), personal communication.

WILLIAMS, T. J. (1938), *Amer. J. Obstet. Gynec.*, vol. 35, page 863.

WYNNE, H. M. N. (1919), *Bull. Johns Hopk. Hosp.*, vol. 30, page 15.

—— (1929), *Amer. J. Surg.*, vol. 32, page 389.

THE LACTATING NODULE*

By E. Hirst, D. L. Glen and R. M. Rawle

Sydney Hospital

THE discovery of a lump in the breast of a pregnant or recently parturient woman may alarm both patient and physician, because of the well known poor prognosis of carcinoma arising in this period (Geschickter, 1943; Haagensen, 1956). Little reassurance is to be found in a study of the standard surgery and pathology texts for, apart from cancer, one may only learn that fibroadenomata may grow rapidly during pregnancy. However failure to elicit a history of a pre-existing lump, or knowledge of its absence from prior examination, tends to confirm the physician's fears. If exploration is performed and the tumour is found either partly or entirely to lack encapsulation, the surgeon may find the lesion difficult to assess and radical surgery may be performed.

In this paper, we report eight instances of a lump in the breast arising during pregnancy, encountered in the course of 259 consecutive breast biopsies submitted for frozen section examination. A report by Cortis (1958) on 100 consecutive lumps in the breast included two of these lesions and these are also included in the present series. In the same period one instance each of carcinoma in pregnancy, inflammatory carcinoma in pregnancy and fibroadenoma in pregnancy were encountered.

Clinical Features

Table 1 shows the main clinical features. With one exception the lump was noted for the first time during pregnancy. In this instance the patient had noticed "a small lump the size of a grain of wheat" two months prior to pregnancy.

The lumps were all painless, and in three instances they were tender. They were discovered by the patient or during routine antenatal examination. There was no instance of nipple discharge. As regards the remainder of the mammary tissue, it was noted in one patient that the other breast "tended to be lumpy." In the remaining cases the breasts were either normal, or their condition evoked no comment. Of the six multiparous patients,

Table 1

CLINICAL FEATURES OF LACTATING NODULE

Case No.	Age	Stage of Pregnancy when Excised	Duration of Symptoms	Parity	Site Quad.	Size cm.	Mobility	Consistency	Remarks
1	22	2 weeks post-partum	5 months	Primip.	Upper Outer	5 × 2	Slight fixation to skin	Hard	Grew rapidly last week Feeding baby at excision
2	24	5 weeks post-partum	11 months	Multip. (3)	Upper inner	1 cm. diam.	Mobile	Rubbery firm	Lactation suppressed, gradual increase in size
3	30	12 weeks	2 weeks	Multip. (1)	Upper inner	4 × 3	Not mobile	Soft	—
4	36	16 weeks post-partum	26 weeks	Multip. (3)	Lower inner	1.5 cms.	? deep fixation	Soft	Not feeding baby
5	17	30 weeks	6 weeks	Primip.	Axillary tail	3 cm.	Skin dimpling	—	—
6	25	36 weeks	32 weeks	Multip. (2)	Lower inner	Small	Mobile	Firm	—
7	24	24 weeks	6 weeks	Multip. (2)	Upper inner	1 cm.	Mobile	Firm	—
8	34	32 weeks	6 weeks	Multip. (1)	Axilla	3 cm.	Fixed to axillary skin	Firm	—

*Received for publication 19th July, 1960.

some had breast-fed their children while others had not. None of the multiparous patients had a lump in any previous pregnancy. In five of the patients the lump was situated in the inner half of the breast. In one patient the nodule was attached to the deep part of the skin of the axilla. In three of the cases the lumps were mobile in the breast tissue. In the remainder the lumps were not mobile in the breast. A slight degree of fixation to the skin only was noted in three cases and possibly to deeper structures in a fourth. All the lumps were solitary. The women were mainly young, as is to be expected. In no instance did the overlying skin show any evidence of inflammation. The axillary lymph nodes were palpable and small in two cases. In all the remaining cases they were not palpable.

(a) (b) (c)

FIG. I. (a) Shows an ill defined nodule; (b) Case 8, shows one of intermediate demarcation; (c) Case 6, a well-defined nodule.

PATHOLOGY

The nodules measured from 2.5 cm. to 4 cm. in maximum extent, four were of the largest size (4 cm.). They had been excised with a variable amount of surrounding breast tissue. In one instance the nodule was virtually shelled out, with only a little surrounding adipose tissue. The degree of demarcation of the nodule from the surrounding breast tissue was variable. In 2 instances it was very well defined (Fig. Ic). However no mobile fibrous capsule was found. In 2 of the nodules the periphery showed some degree of delimitation from the surrounding breast tissue (Fig. Ib). One of these was the nodule attached to the skin of the axilla. The 4 remaining nodules had an irregular outline and lacked, except in an occasional area, demarcation from the surrounding breast (Fig. Ia). The cut surface in 5 of the nodules was pale brown, in one pale pink and in 2 greyish-yellow. Close inspection showed a glandular pattern. Fixation in formalin tended to reduce the brown colour.

Microscopic examination of the nodules showed that they all had a lobular pattern with development of acini as found in the breast in pregnancy. All were undergoing secretory activity as evidenced by the presence of secretion in the acini and of fat globules in their lining cells. In advanced pregnancy the lobules were large (Fig. II) but the lobular pattern was present. The degree of advancement of pregnancy changes was assessed by the density and size of the lobules, the density of the acini developing in them and by the accumulation of secretion in the acini. Thus of the 5 nodules excised during pregnancy, the degree of development of pregnancy changes in the nodules was more advanced where pregnancy also was more advanced. Ducts were present in all the nodules and in about half the cases were slightly dilated and contained a little secretion. In the remaining cases the ducts were small and empty. The lobules were separated from each other by a variable amount of fibrous connective tissue, dependent upon the size and density of the lobules. Thus the amount of interlobular connective tissue was small in those nodules where pregnancy was far advanced. No adipose tissue was found in the nodules. In only one nodule was any inflammation found. It consisted of a zone of chronic inflammation

(mainly lymphocytes) surrounding a small area of dilated ducts and acini which contained inspissated secretion. This was interpreted as a small focus of spilt secretion mastitis and was absent from all other nodules and from the surrounding mammary tissue.

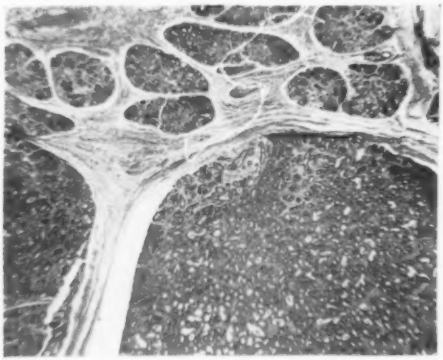


FIG. II. Case 5. Histological appearances of the junction of a well-defined nodule. Advanced secretory changes in the lobule and less marked changes in surrounding mammary tissue (at top). (x 54)

In comparing the degree of advancement of the pregnancy changes in the nodules with that in the surrounding breast, it was found that in the earliest case (Case 3, Fig. III) the nodule at twelve weeks showed a well-marked lobular pattern. The surrounding breast however showed only proliferation and budding of the ducts which were surrounded by characteristic oedematous connective tissue. At twenty-four weeks gestation (Case 7, Figs. IV and V) the changes were similar. At thirty weeks gestation however (Case 5, Fig. II) the changes both in the nodule and in the surrounding breast were more advanced. It is of interest to note that the lobular development in the surrounding breast had now reached the stage seen in the nodule in the earlier cases. The changes at thirty-six weeks gestation were similar to those at thirty weeks. The findings in the nodules excised postpartum are interesting. Only one of these patients was feeding her child. This nodule showed advanced secretory changes and multiple areas of necrosis (Case 1, Fig. VII). Coagulative necrosis was also present in one of the lesions reported by Gill, Stirman and Gordon (1953). In the other 2 nodules, the patients were not feeding their children (in one case lactation had been suppressed) and the degree of lobular density and development was similar to that seen in the nodules of early pregnancy. In these 2 cases however the surrounding breast tissue contained small widely separated lobules, rather than proliferating ducts. These 3 cases showed the same features as those in which the nodules were excised during pregnancy, namely an advanced degree of pregnancy change in the nodules compared with the surrounding breast. In the nodule attached to the skin of the axilla (Case 8, Figs. VIII and IX) pregnancy changes were not as advanced as in the nodules excised from the breast proper at a comparable stage of pregnancy.

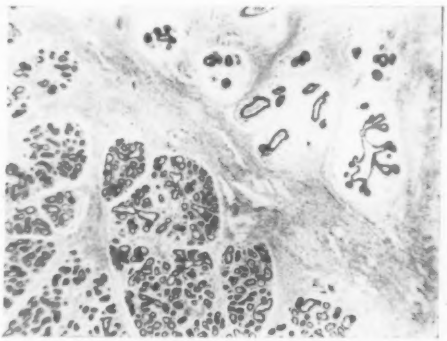


FIG. III. Case 3. Nodule early in pregnancy, showing junction of nodule with surrounding breast (top right). (x 54)

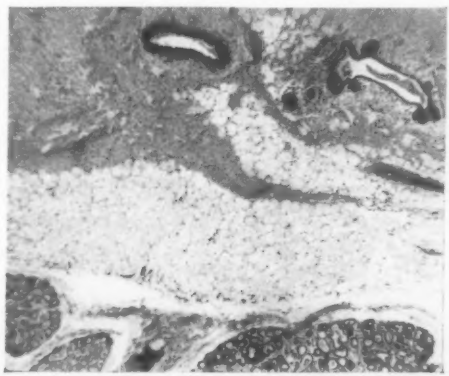


FIG. IV. Case 6. Junction zone consists of adipose tissue. (x 54)

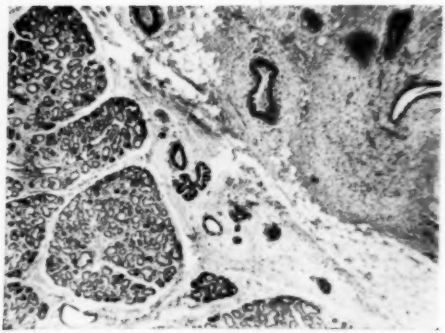


FIG. V. Case 6. Interdigitation of nodule and surrounding breast. Nodule early in pregnancy. (x 54).

The periphery of the nodules was studied microscopically in order to determine the nature of the junction between the nodule and the surroundnig breast. This junction was found to be variable in each nodule and in each case. Where the nodule was macroscopically well-defined, in some areas a zone of adipose tissue separated the nodule from the surrounding breast (Fig. IV). Even in these nodules however, this zone of adipose tissue was broken up by areas of developing breast tissue which interdigitated with the more advanced lobules of the lactating nodule (Fig. V). In other areas a junctional zone consisted of fibrous connective tissue (Fig. III). In the case of the nodule which was shelled out, a surrounding zone of adipose tissue contained smaller, less well-developed lobules (Fig. VI). In many parts of the well-defined nodules there was no sharp junctional zone between the nodule and the surrounding breast. In the poorly defined nodules such areas of interdigitation were the rule. In no instance was a capsule in the form of an envelope of areolar tissue found. In the axillary nodule attached to the skin (Case 8) the surrounding tissue contained apocrine and eccrine sweat glands interdigitating with the developing breast tissue of the nodule (Fig. IX). Although this case may be termed polymastia, the nodule arose during pregnancy in a multipara and appears to be the same as the nodules occurring in the breast proper.

These findings indicate that lactating nodules consisted of breast tissue in a more advanced stage of pregnancy change than the surrounding breast and that they maintained this lead as pregnancy advanced. In the case of the nodules excised post-partum where the child was not being fed, they apparently retained their secretory development in the face of involutionary changes in the surrounding mammary tissue.

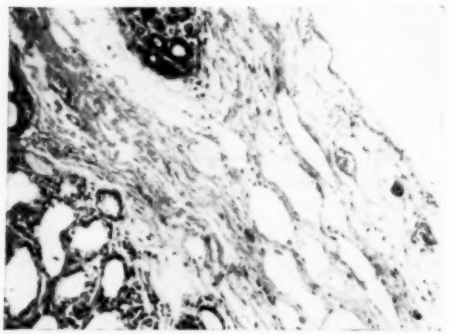


FIG. VI. Case 1. Lactational nodule showing periphery of adipose tissue in which there is a small lobule with less advanced pregnancy change. (x 213)

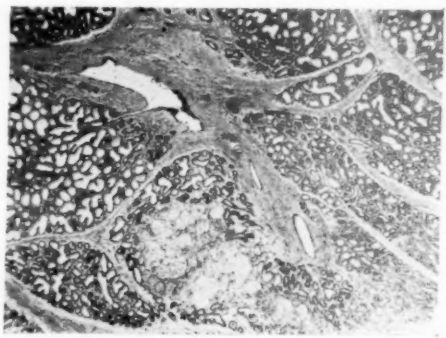


FIG. VII. Case 1. Lactational nodule. Very advanced secretory changes and areas of necrosis. (x 54)

No similar lesion has been found in a patient who was not either pregnant or lactating.

LITERATURE

Little has been written during the last ten years in the English literature regarding lumps in the breast during pregnancy. Moreover the major papers published previous to

this are difficult to assess since they do not separate lumps arising during pregnancy from those previously present.

Of the papers reviewed, only that by Gill, Stirman and Gordon (1953) deals exclusively with a lump in the breast appearing for the first time during pregnancy or lactation. Under the title "Lactating Adenomas of the Breast" these authors describe 6 cases, all in multiparae, the oldest patient being 34 years of age. It is of special interest to note that in 2 cases the lumps appeared during a previous pregnancy. The nodules were firm or hard and in 5 of their patients, were situated

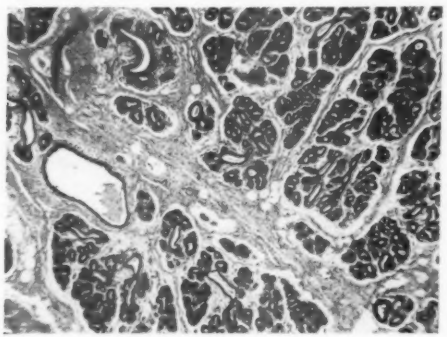


FIG. VIII. Case 8. Axillary nodule. Developing breast lobules. (x 54)

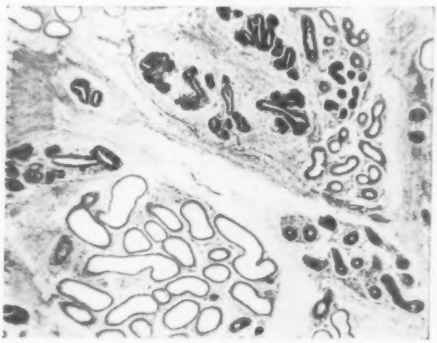


FIG. IX. Case 8. Axillary nodule. Periphery of nodule. (x 54)

in the inner half of the breast. Macroscopically, they gave the impression of being well circumscribed but were not sharply encapsulated. The histological features were similar to the usual lactating or pre-lactating breast tissue. The authors state that the clinical and pathological features are such that it should be possible to include the lesion in the list of pre-operative diagnoses and to make the correct diagnosis in many.

However Cheatle and Cutler (1953) describe "lactating adenomas" as pre-existing slowly growing benign tumours that suddenly begin to enlarge during pregnancy and often grow more rapidly as lactation commences. Though emphasizing these features as characteristic of the history and clinical course they refer by way of illustration (their Case 3, Fig. 430) to a tumour which had appeared as a painless lump when the patient was five weeks pregnant; it was biopsied several times and continued to grow. Eventually simple mastectomy was performed. In their next case the patient had a lump which was stationary for years and commenced to grow during pregnancy.

Hill and Miller (1954) report 8 cases of "adenoma" of the breast. Six of their patients were either pregnant or lactating and of these the lump appeared in the medial half of the breast in at least 3 cases. Their oldest patient was 40 years of age. All the tumours were "usually partly or completely encapsulated" and they usually showed a tan coloured cut surface "similar to a lobule of lactating breast." These authors stressed the presence of encapsulation and ease of enucleation in differentiating adenomas from focal hyperplasias. They considered an adenoma as a "purely glandular tumour where connective tissue took no part in the neoplastic process."

The possibility that tumours arising in the breast during pregnancy are modified fibroadenomas has been canvassed in the older literature. Thus Moran (1935) describes 27 cases of fibroadenomata during pregnancy and lactation. Many of these lumps arose during the present pregnancy while 6 were of longer standing. There is only one photomicrograph of the long-standing lumps which does not resemble the lesion herein described. The remainder of the illustrations show either a lesion similar to our lactating nodule, or an area of pregnancy hyperplasia merging into fat, or are not relevant. Since no characteristic fibroadenoma is illustrated it may be that these long-standing cases were unmodified

by pregnancy change. There seems to be no doubt that some of the lesions described by this author are similar to those that we now describe. Moran notes that in 15 cases of this series the lesion was confused with cancer.

Geschickter and Lewis (1938) have also described pregnancy and lactational changes in fibroadenomata of the breast. In 10 of their patients the lump appeared during pregnancy, was macroscopically well demarcated from the rest of the breast but microscopically they showed "non-encapsulation at one or more points." These authors also noted a tendency of the tumour "at first to exceed and later lag behind the physiological development of the breast." They also noted that tumours of long-standing may be refractory pregnancy changes. In their series of 13 cases, though a few cases represented pre-existing tumours refractory to pregnancy change, others showed the clinical, gross and microscopical characteristics similar to the nodules in our series.

Geschickter (1943) states that fibroadenomata may grow rapidly during pregnancy and that despite this rapid growth remain encapsulated and freely movable. He describes a fibroadenoma in pregnancy as a pre-existing, firm, circumscribed breast nodule which had remained stationary in size. Rapid growth during pregnancy is often sufficiently characteristic to permit a clinical diagnosis of both conditions, fibroadenoma and pregnancy.

In Bloodgood's paper (1924) a nodule showing the histological appearances of lactating nodule is depicted. He records encountering non-encapsulated lumps in the breast which could not be shelled out. He advised excision of the breast but stated that later study showed many of these lesions to be benign. The state of the literature on these breast nodules can be illustrated by the contradictory captions for photomicrographs given by Bloodgood (his Fig. 21) and by Moran (his Fig. 8). These photomicrographs are identical and must have come from the same lesion, yet Bloodgood states that the nodule was removed at the end of lactation while Moran says it was removed at the second month of pregnancy.

In another instance Bloodgood (1924) depicts a photomicrograph (his Fig. 20) as showing "lactation hypertrophy in an encapsulated fibroadenoma removed during lactation." However Oliver and Major (1934) show the identical photomicrograph (their Fig. 36) stated to be from a women six months pregnant and regard it as noteworthy that "adjacent to this marked epithelial hyperplasia seen in the illustration, there are lobules which contain few parenchymal elements and much loose connective tissue." In our view the photomicrograph is typical of that of a lactating nodule as reported herein and it is of interest that the nodule was situated in the medial half of the breast.

DISCUSSION

In considering the pathological nature of a lump in the breast arising during pregnancy or lactation other than carcinoma, the above possibilities as well as hyperplastic cystic disease and asynchronous response of portion of the breast should be taken into account. There is evidence that pre-existing fibroadenomata may undergo pregnancy and lactational change, though little of it is pictorial or convincing. Nicholson (1921) has described a fibroadenoma with pregnancy changes in which a clearly defined capsule in the form of an envelope of loose areolar tissue surrounded the tumour. Other authors fail to show convincing evidence of a capsule in many of their nodules which they often describe as "more or less encapsulated." Hill and Miller (1954) express doubt that changes present in their cases had been shown to occur in a fibroadenoma modified by pregnancy. They state moreover that a fibroadenoma in a pregnant women studied by them showed no modification. Our experience has been the same, in that a fibroadenoma in a gestational breast showed no pregnancy change. We consider our lesions to be the same as those depicted by Hill and Miller. The characteristic criteria for fibroadenoma in pregnancy are pre-existing tumour, increase of growth during pregnancy, free mobility and encapsulation. This is in contrast with our patients in whom the tumour arose during pregnancy and showed no encapsulation (though some degree of demarcation from the surrounding breast tissue).

The histological features of lactating nodules do not resemble those of a fibroadenoma in that there is always a well marked lobular pattern and ducts are present in the interlobular connective tissue. Fig. VII depicts a communication between a lobule and duct and indicates that the structure is essentially that of normal breast. The characteristic pericanalicular or intracanalicular pattern of a fibroadenoma has not been found in any part of our nodules nor has any instance been depicted in the literature reviewed.

Other features suggesting that these nodules are not lactating fibroadenomata are the absence of changes in the surrounding breast tissue and their position in the breast. Willis (1953) has pointed out that foci of hyperplastic cystic disease and of fibroadenomatosis are often present in breasts that contain a fibroadenoma. These features were absent from the surrounding breast in our cases. The predominance of lactating nodules in the inner half of the breast (Gill, Stirman and Gordon, 1953) in our cases and possibly in those of the pregnant women in Hill and Miller's series, contrasts with the predominant situation of fibroadenomata in the upper and outer quadrant (Oliver and Major, 1934). In maintaining that the lesions are true neoplasms (adenomata) in which "only the glandular component takes part in the neoplastic process" Hill and Miller (1954) exclude the possibility of a focal hyperplasia by the presence of "encapsulation," ease of enucleation and distortion of the architectural pattern compared with the remainder of the breast. In most of our patients and in those of Gill, Stirman and Gordon (1953) these features were absent. Moreover in Hill and Miller's illustration there appear to be changes of pregnancy in the lesion in advance of those of the surrounding breast, a feature characteristic of our cases. Willis (1950) considers it doubtful if a pure adenoma ever occurs while Geschickter (1943) states that "apart from rare cases in your girls where the epithelial component predominates, the remainder of pure adenomas are intracystic papillomas."

Gill, Stirman and Gordon (1953) title their paper "Lactating Adenomas of the Breast" but favour the possibility that the lesions are nodules of breast tissue without normal connections with the rest of the breast. It is significant that they do not describe dilatation of the ducts or accumulation of secretion and ensuing inflammatory changes. These features are also absent from the nodules in our patients other than a microscopical area in one nodule.

Broders, in the discussion of the paper by Gill, Stirman and Gordon, suggested that the term "lactating adenosis" be used for these nodules. The implication is that changes of hyperplastic cystic disease may be found in the remainder of the breast tissue. Of our patients, only one showed clinical features suggestive of hyperplastic cystic disease and this was not confirmed on histological examination of the surrounding mammary tissue.

To draw attention to a lump arising in the breast during pregnancy or lactation and which at operation may well be demarcated from the surrounding breast tissue, or may show partial or entire lack of definition from it, we prefer to use the term lactating nodule. We feel however that our evidence shows that it is probably an asynchronous response of portion of the mammary tissue to the hormonal stimuli of pregnancy or lactation. A similar view has been advanced by Oliver and Major (1934). Cheatle and Cutler (1931) indicate that a different response in various parts of the breast is a common finding in pregnancy and illustrate it convincingly (their Fig. 17). Geschickter (1943) confirms this finding and illustrates (his Fig. 33) marked irregularity in physiological changes in different parts of the breast. This illustration corresponds closely to the changes found by us in lactating nodules.

PROGNOSIS AND MANAGEMENT

The treatment in the present series has consisted of resection of the lesion with a little surrounding breast. Though follow-up is short, there have been no instances of recurrence. Two patients became pregnant, respectively two and three years after excision of the nodule. The lesion did not recur.

Assuming that the lesions discussed above correspond to our lactating nodule, there has been no recurrence in papers reviewed. In Oliver and Major's (1934) case, treatment was by excision with no recurrence after eleven years. In the case quoted by Cheatle

and Cutler (1931) a nodule continued to grow during pregnancy and the breast was excised. In 2 cases (Gill, Stirman and Gordon) a lump had appeared in a previous pregnancy. On the other hand in 2 cases (Moran, 1935) excision eight years after showed "cystic disease."

The clinical and gross characteristics have been such in our series that one of us (R.M.R.) has been able to suggest the diagnosis pre-operatively and recognize the lesion grossly at operation.

Nevertheless we feel that excisional biopsy would normally be indicated because of the uncertainty related to such nodules. There are 2 instances reported (Geschickter and Lewis, 1938; Hill and Miller, 1954) of carcinoma associated with an "adenoma."

According to Hill and Miller (1954) the appearances of these nodules on frozen section may be disconcerting. In our experience however fresh frozen section greatly facilitates the recognition of these nodules. The globules of fat are readily visible in the cells lining the acini, and in no instance has the diagnosis of malignancy or an equivocal report been given.

SUMMARY

1. In 8 women a lump arose in the breast or axilla during pregnancy or lactation. It was excised to exclude carcinoma and did not recur in subsequent pregnancies.
2. The lumps occurred predominantly in the inner half of the breast and were of consistency varying from soft to hard. They were single, painless, usually non-tender, three were mobile in the breast, the remainder partly fixed.
3. Macroscopically they were characterized by variable demarcation from the surrounding breast tissue and although several could have been shelled out, none showed a true capsule. Their cut surface was mainly pale brown and a glandular pattern was usually visible.
4. Histologically they showed a lobular pattern with pregnancy and lactational changes in advance of those of the surrounding tissue.
5. Since their histogenesis is uncertain we suggest the term lactating nodule.
6. A possible explanation lies in the asynchronous response of portion of the breast to a hormonal stimulus.

ACKNOWLEDGEMENTS

We wish to thank Dr. A. A. Palmer, Director of Clinical Pathology, for valuable help and suggestion; Mr. C. E. Winston, Honorary Consulting Surgeon, and Mr. J. V. L. Colman, Honorary Assistant Surgeon, for permission to study lesions and clinical notes from their patients, and Miss Sylvia Wilkins for photographs.

REFERENCES

BLOODGOOD, J. C. (1924), *Ann. Surg.*, vol. 79, page 172.

BOTHE, A. E. (1925), *Amer. J. med. Sci.*, vol. 170, page 731.

CHEATLE, G. L. and CUTLER, M. (1931), "Tumours of the Breast." London, Edward Arnold & Co.

CORTIS, E. M. (1958), *Med. J. Aust.*, vol. 1, page 668.

GESCHICKTER, C. F. (1943), "Diseases of the Breast." Philadelphia, J. B. Lippincott Co.

—— and LEWIS, D. (1938), *Brit. med. J.*, vol. 1, page 499.

GILL, A., STIRMAN, J. A. and GORDON, C. E. (1953), *Tex. St. J. Med.*, vol. 49, page 231.

HAAGENSEN, C. D. (1956), "Diseases of the Breast." Philadelphia and London, W. B. Saunders.

HILL, R. P. and MILLER, F. N., Jr. (1954), *Cancer*, vol. 7, page 318.

MORAN, C. S. (1935), *Arch. Surg.*, vol. 31, page 688.

NICHOLSON, G. W. (1921), *Guy's Hosp. Rep.*, vol. 21, page 1.

OLIVER, A. L. and MAJOR, R. C. (1934), *Amer. J. Cancer.*, vol. 21, page 1.

WILLIS, R. A. (1950), "The Principles of Pathology." London, Butterworth Co.

—— (1953), "Pathology of Tumours." London, Butterworth Co.

THE LATE SEQUELAE FROM INJURIES TO THE BRACHIAL ARTERY: THEIR MANAGEMENT*

By R. P. Jepson and L. J. Opit

University Department of Surgery, Queen Elizabeth Hospital, Woodville, South Australia

THE management of an arterial injury depends considerably on its site and the condition under which it is inflicted. Acute traumatic lesions have a deservedly poor reputation. As reported by DeBakey and Simeone, 601 gunshot wound injuries to the brachial artery in the U.S. Forces during World War II were followed in 159 cases (26.5 per cent.) by the loss of the limb. In contrast it is known that non-traumatic brachial, axillary or subclavian artery thrombosis carries a benign prognosis and is rarely accompanied by severe nutritional changes (Jepson, 1957). The reason for this divergent outcome in the two groups is probably explained by the fact that traumatic injuries are more than simple interruptions of the main limb artery. Profound circulatory upset occurs through multiple injuries, blood loss, hypotension, gross accompanying bone and soft tissue damage impeding collateral circulation, venous damage and peripheral oedema. The biochemical changes of the stress response may also deleteriously interfere with muscle metabolism. The management of such complicated vascular injuries is an urgent problem which has recently been reviewed by Hardy and Tibbs (1960) and will not be considered further.

In this paper we present three cases where the brachial artery was acutely interrupted by simple incisional or contusional injuries without any severe constitutional upset. The viability of the limb was never in jeopardy and the indications for surgical intervention in two of these were those of functional incapacitation brought about through vasospastic phenomenon or muscle claudication.

Case 1

F.F.H., male, aged 31 years, was a rabbit trapper. On 20th December, 1959, his car overturned contusing his right shoulder and upper arm and left leg. The most important injury was that to the right arm where he sustained a deep laceration just below the mid-humerus on the inner side. This caused two lesions, a median nerve lesion which was almost complete and division of the brachial artery at the same level. Moderate bleeding resulted which was readily controlled by bandaging. When seen two months after the injury there was patchy hypoaesthesia present in the median nerve distribution, there was movement in the long flexor to the middle finger, otherwise the interruption of the median motor supply was complete. He had noticed since the day of the accident that the hand, in addition to being numb and weak in the median nerve distribution, was also colder than the left hand. On examination there was evidence of muscle wasting in the forearm (but not muscle infarction) and although the right hand was a few degrees colder than the left at room temperature, a weak radial pulse, noticed to be absent immediately after the accident, had returned. Electromyogram and sweat test confirmed the severe median nerve injury.

On 16th February, 1960, the scar over the inner side of the upper arm was excised under general anaesthesia and the lesions explored. The main bulk of the median nerve was anatomically intact, although in its lateral aspect a small laceration was adherent to the periosteum. The nerve was dissected free of scar tissue. The brachial artery had been divided by the accident at the same level, the ends having separated about 1" and retracted into an intact adventitial casing. In view of the return of pulse and localized nature of the arterial injury not involving any large collateral vessels, it was decided not to perform a graft or ganglionectomy. When seen one month later in the out-patient department the skin of the fingers supplied by the median nerve was sweating, sensation was returning and the radial pulse had become stronger.

Comment

The major disability in this patient was the partial median nerve lesion. Within a month or so of the injury the radial pulse was readily palpable and the patient asymptomatic from a vascular viewpoint. If after nerve recovery forearm claudication results on heavy exercise, he will be re-admitted for by-pass grafting. Retraction of the divided vessel ends within an intact sheath of adventitia has sometimes been wrongly interpreted as "vascular spasm" (Hardy and Tibbs, 1960) and is the explanation of the minimal bleeding which occurred with complete transection of the vessel.

*Received for publication 26th July, 1960.

Case 2

H.E.N., male, aged 52 years, was a mechanic. On 18th December, 1959, while at work he closed the back door of a van with a push by his outstretched right arm. Within a few minutes the forearm and hand became "dead" and "numb." This passed away after a few days but in the following three months the patient noticed (a) that the fingers became white on exposure to cold, particularly if the hand was used as for wringing clothes; (b) an ache in the forearm on exercise such as that involved with a hammer, or pushing a lawn-mower. This patient had no previous history of vascular impairment. He had undergone a partial gastrectomy in 1947 for a duodenal ulcer and he suffered from attacks of weakness and faintness since being a prisoner of war in Poland in 1942.

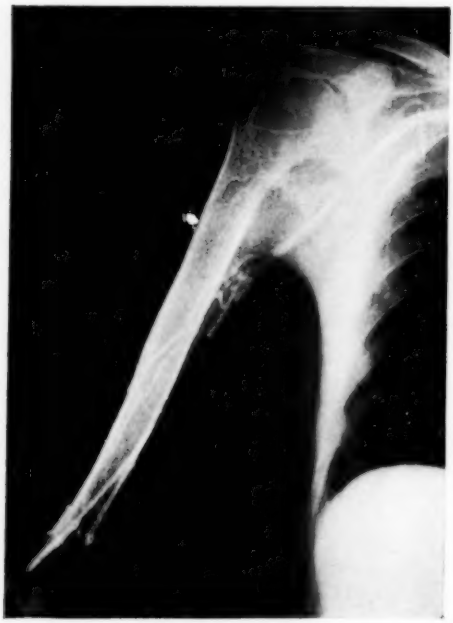


FIG. 1 Case 2. Pre-operative arteriogram showing thrombosis of mid-humeral segment of brachial artery.

On examination the patient was normotensive (blood pressure 120/80 mm. of mercury in the left arm) with no evidence of a vascular defect except in the right arm where the brachial artery was palpably thrombosed over a region of 3 to 4 cm. at the level of the insertion of coracobrachialis. The right radial pulse was very weak, scarcely palpable and disappeared after mild exercise of the arm and on elevation. The fingers of the right hand were cold and could be provoked into Raynaud's phenomenon by immersion in cold water. No source for embolism could be found (E.C.G. normal) and neither clinically nor radiologically was there evidence of a cervical rib.

An arteriogram (Fig. 1) was performed on 4th March, 1960, which confirmed the clinical diagnosis of a thrombosis of the brachial artery opposite the middle third of the humerus. The vessels, both proximal and distal to the thrombosis, were perfectly smooth and did not appear to be involved in any degenerative process.

As occlusive involvement of the brachial artery is extremely uncommon in atherosclerosis and as the patient had no evidence elsewhere of arterial degeneration, it was presumed that the thrombosis was attributable to the closing of the van door which immediately preceded the thrombosis. In view of the distressing symptoms which persisted for three months, it was decided to explore the lesion with a view to inserting a graft.

On 11th April, 1960, an axial incision was made over the neurovascular bundle exposing the brachial artery from axillary fold to 4 cm. above the antecubital crease. Normal artery was found proximally with large muscular collaterals and patent profunda: there was no evidence of atherosclerosis to the naked eye or to palpation. At about the mid-humerus level a 3 to 4 cm. clot with some broadening of vessel diameter was located. A reversed vein graft of 10 cm. (5/0 continuous suture) was employed as a bypass. The only technical difficulty was the small size of the collapsed brachial artery distal to the clot. The radial pulse had returned to the equivalent of the left within twenty-four hours of operation. In view of the vasospastic nature of some of the preoperative symptoms a thoracic ganglionectomy (excision of first, second and third thoracic ganglia with the intervening chain) was performed at the time of the grafting procedure.

Comment

The mechanism of this thrombosis is not clear. There is no evidence of segmental or widespread atherosclerosis in this man. Possibly the action of closing a door was sufficient to cause a "stretch-lesion" at the site of a muscular branch. Possibly the true diagnosis will only be reached on long-term follow-up.

Case 3

W.E.S., male, aged 53 years, was a moulder. On 29th March, 1960, while working underneath a raised-up Goggomobile the jack slipped causing the underneath edge of the body to crush the inner side of the right arm against the floor. A large bruise was immediately evident and the hand became cold, blue and numb. An X-ray showed no associated fracture. He remained at work in the succeeding days while noticing (a) the forearm swelled and the fingers tingled after using the arm; (b) hammering or screwing caused forearm claudication after a few minutes, this pain being characteristically relieved by resting for two or three minutes; (c) the hand remained colder than the left side and if exposed to cold immediately became cyanotic but true Raynaud's phenomena were not evident.

On examination 3rd May, the patient was hypertensive (170/130 mm. of mercury in left arm), otherwise all abnormalities were restricted to the

right arm. There was some light cyanosis in the right hand, detectable wasting of forearm muscles, a faintly palpable pulse at right wrist and a barely palpable brachial pulse in the antecubital fossa. The upper third of the brachial artery below the axilla was readily and normally palpable; however, below this the artery was thrombosed and a thickened segment, running down the arm for 3 or 4 cm. was felt in the line of the brachial vessels. An arteriogram (Fig. II) showed a complete 5 cm. occlusion of the brachial artery just below the origin of the superior ulnar collateral with refilling of the artery below. In view of the claudication in forearm muscles it was decided to insert a free vein graft.

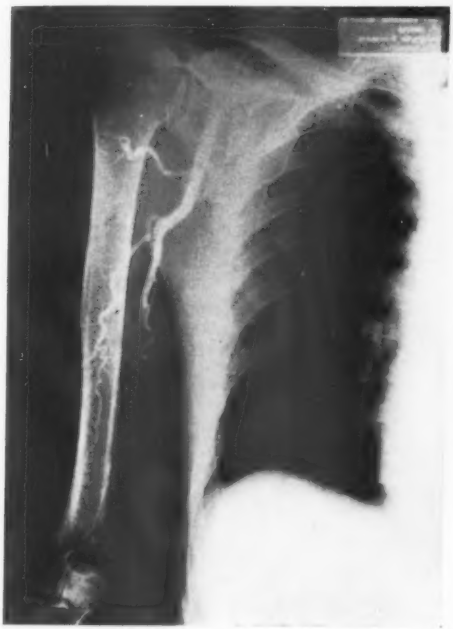


FIG. II. Case 3. Pre-operative arteriogram showing thrombosis of mid-humeral segment of brachial artery. Refilling of brachial artery beyond occlusions was seen in further films in this series.

On 10th May, 1960, the thrombosed segment was exposed by an axial incision to expose 5 to 6 cm. of normal artery above and below. The occluded vessel was about twice normal diameter and solid with clot, having the greenish hue of a recent thrombosis and considerable perivascular adhesions. Segments of normal artery were dissected out above and below; the later artery was a half the diameter of that above the thrombosis. Temporary ligations were applied and a 10 cm. piece of internal saphenous vein inserted to by-pass the occlusion. Post-operatively the radial pulse returned forcibly and the arm has remained asymptomatic since operation (Fig. III).

Comment

The sole indication for operation was that claudication proved to be an economic handicap. As in analogous lesions of the leg, it is unlikely that collateral flow without grafting would ever be sufficient to sustain heavy and continuous exercise.

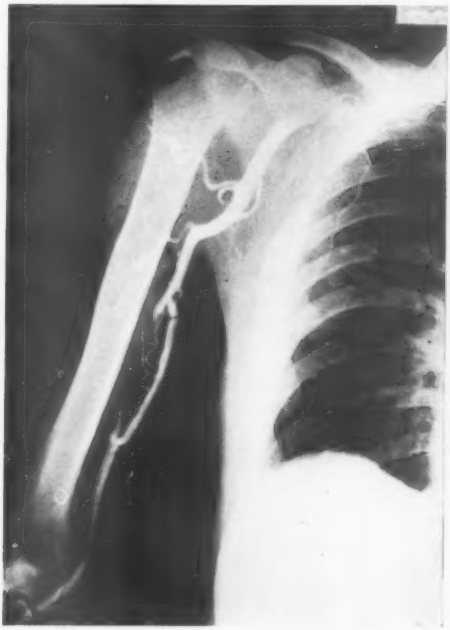


FIG. III. Case 3. Post-operative arteriogram: the reduction in size of the proximal anastomotic collaterals is evident now the main channel has been reconstituted.

DISCUSSION

The cases discussed herein are problems in rehabilitation rather than the salvaging of acutely ischaemic limbs. In general we believe (Jepson, 1955) that most acute non-traumatic occlusive lesions of the upper arm vessels can be treated conservatively with confident survival of the limb and minimal functional disability. As the majority of such vascular episodes occur in the elderly or those incapacitated by cardiac or peripheral vascular disease elsewhere, no attempt at direct surgery is required, although on occasions a cervico-thoracic ganglionectomy may be required for impaired hand and finger circulation. In Cases 2 and 3 (and possibly in

Case 1 when the median nerve recovery has taken place) the patient was handicapped by forearm muscle ache on exercise and in Case 2 with vasospastic attacks in the fingers. The circulatory demands of muscle on exercise are such as to make direct surgery and the reconstitution of a major arterial channel the only feasible means of relief. For this a by-pass operation has several distinct advantages as it enables dissection to be limited to normal tissues only, well away from the occluded area and it allows a reasonably large oblique anastomosis to be made in the collapsed vessel distal to the clot. For the purpose of the graft, internal saphenous vein has much to recommend it. It is a viable graft: the "average" internal saphenous vein is of the correct diameter; it is easy to work and fit into the arterial bed. Our impression, from the use of autogenous vein grafts in the legs, is that autogenous vein graft has the best immediate and long-term patency rate when compared with synthetics or preserved homologous artery.

One or two technical points are of some importance. The vein graft when removed from the thigh tends to shrink and contract. To counter this a temporary occlusion is placed on one end and the vein is distended (overstretched) with an isotonic saline injection. Its ends are cut cleanly and obliquely. The arterial adventitia, which is a tough, easily defined structure, must be cut away from the anastomotic line before a suture is commenced. Following this ellipses (2 mm. by $\frac{3}{4}$ cm.) are cut out of the artery at the graft sites. It is our preference to use a continuous 5/0 silk over and over closure. Under arterial pressure the graft will arterialize and lengthen and therefore, to avoid undue tortuosity the graft should be inserted under fair tension. It is not necessary to stress that the graft should be inserted in such a direction that the valves do not impede flow.

SUMMARY

1. Three cases of traumatic brachial artery interruption are reported.
2. In two of these a free vein graft was inserted because of symptoms severe enough to interfere with gainful occupation.
3. Some technical points concerning vein graft insertion are mentioned.

ACKNOWLEDGEMENTS

Our thanks are due to Mr. J. D. Sidey and Dr. R. F. West for referring cases to us for treatment.

REFERENCES

DEBAKEY, M. E. and SIMEONE, F. D. (1955), "Vascular Surgery in World War II," edited by Elkin, D. C. and DeBakey, M. E., Office of the Surgeon General, Dept. of the Army, Washington, D.C.

HARDY, E. G. and TIBBS, D. J. (1960), *Brit. med. J.*, vol. 1, page 1001.

JEPSON, R. P. (1957), *Ann. roy. Coll. Surg. Engl.*, vol. 20, page 249.

PERIPHERAL BLOOD FLOW DURING ANAESTHESIA*†

By G. M. Davidson

Sydney

IT is generally accepted that general anaesthesia produces peripheral dilatation due to loss of tone of the vasomotor centre. Using finger plethysmographs Foster *et alii* (1945) demonstrated an increased blood flow through the fingers and toes during ether anaesthesia and Robertson and Swan (1957), in a summary of available evidence supported this and added that the vasomotor tone returns in about one hour, resulting in a normal peripheral blood flow. Other workers using venous occlusion plethysmography of the forearm, have stated that there is also a vasodilatation in the forearm muscle vessels even greater than in forearm skin during anaesthesia.

Evidence is presented here to show that the vessels of the palmar, plantar and facial skin react to the effects of general anaesthesia in an entirely different way to the vessels of the remaining skin, and that dilatation of muscle blood vessels does not occur during general anaesthesia.

Method

This work was carried out on 47 patients who received a general anaesthetic during routine operations. In each case, needle or wire thermocouple leads were placed into the skin and muscles of the patient and the tissue temperatures were measured on an Ellab universal electric thermometer with ten channels, using the oesophageal temperature as a measure of central body temperature. Provided the external temperature remains reasonably constant and at a lower temperature than the central blood temperature, then changes in skin temperature are an index of changes in blood flow through that area of skin. Accurate quantitative values of blood flow cannot be obtained by such simple measurements, but the various temperature gradients produced in these experiments were such that quite definite conclusions could be drawn from the temperature charts.

Results

Eight representative charts are reproduced below to illustrate the main findings (Figs. I-VIII).

Owing to the limited number of thermocouple leads it was not possible to measure the temperature of all areas on each case, so that summaries only of the mean values of the changes in temperatures of the various tissues and the number of times that each area was used for readings are presented in Tables 1-4. The room temperature remained between 23° and 25° Centigrade throughout these experiments.

Cutaneous blood flow

The graphs and tables showed that there was a significant increase in the blood flow through the palmar, plantar and facial (two cases only) skin following the induction of anaesthesia, and that this vasodilatation persisted provided anaesthesia remained reasonably deep. If excess blood loss occurred, then vasomotor tone returned and the blood flow through these areas fell again. This method did not give any quantitative values for the blood flow but as the skin temperature in these regions reached a level very close to the central body or blood temperature for prolonged periods, then it can be assumed that near maximal vasodilatation occurred in these areas of skin. On awakening from the effects of the anaesthetic there was an equally dramatic fall of temperature and of blood flow through the palmar, plantar and aural skin.

*Received for publication 15th October, 1960.

†This work was carried out while holding a Research Fellowship in Anaesthetics supported by the New South Wales State Cancer Council and the Australian Society of Anaesthetists.

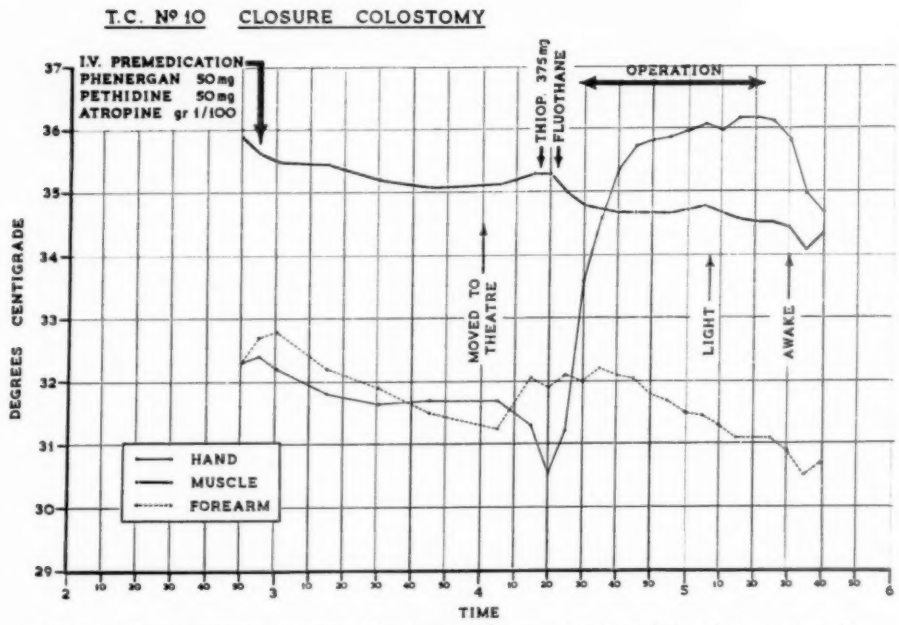


FIG. I. Palmar temperature changed insignificantly after premedication and showed a very steep rise on induction of anaesthesia and a fall on recovery. Forearm temperature did not follow the same pattern. Muscle temperature fell slightly following intravenous premedication and fell more steeply after induction of anaesthesia.

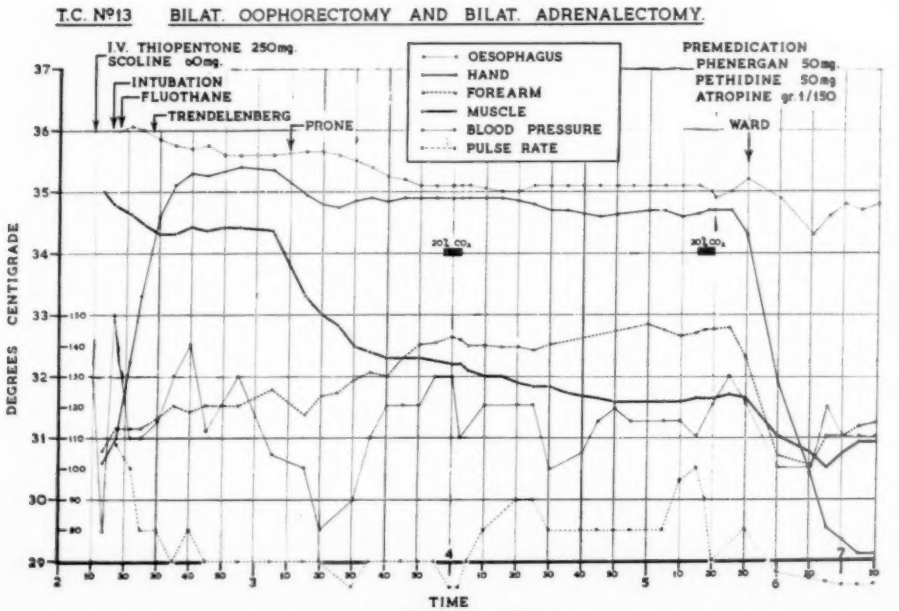


FIG. II. See foot of opposite page.

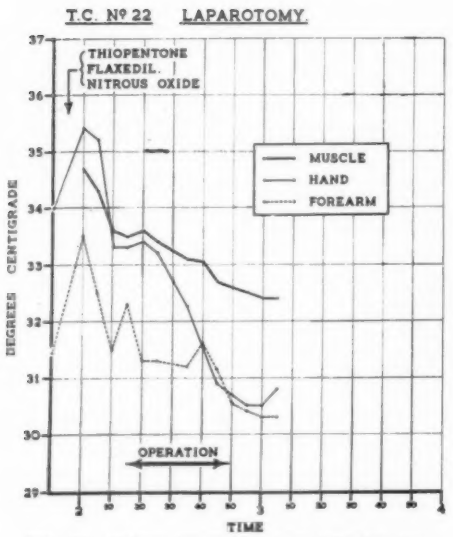


FIG. III. Palmar temperature rose following induction and fell before the operation commenced. This patient did in fact regain consciousness before the operation commenced and remembered most of the ordeal from start to finish except for a few periods of sleep following the subsequent increments of thiopentone. There was minimal blood loss. The forearm temperature followed the palmar temperature at a lower level. The muscle temperature fell.

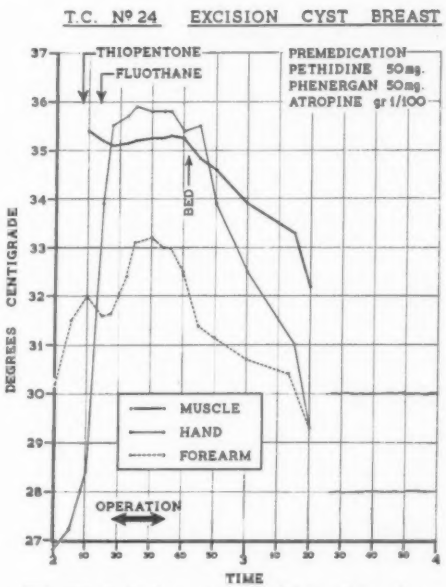


FIG. IV. Induction was followed by a sharp rise in palmar temperature, a moderate rise in forearm temperature and a fall in muscle temperature. Recovery was followed by a fall in palmar and forearm temperature without a rise of muscle temperature.

The pattern of temperature changes of the skin of the forearm and trunk as shown by the graphs and the much lower mean changes as seen in Tables 1-4, indicated that vasodilatation does not always follow the induction of general anaesthesia in these areas of skin and that when it does occur it is far short of maximal as the temperatures reached in these regions were several degrees below the central body temperature as well as being below the palmar temperature. Also, in those cases that were followed for a prolonged period after withdrawing the anaesthetic agents, the forearm and trunk temperatures rose quite rapidly above the palmar temperature towards the central body temperature, indicating that general body heating causes a greater vasodilatation than general anaesthesia in the cutaneous vessels of the forearm and trunk.

All the general anaesthetic agents that were used, namely thiopentone, nitrous oxide, cyclopropane and halothane, showed the same pattern.

The difference between the behaviour of the cutaneous vessels of these two groups of areas was especially noticeable after the discontinuance of the anaesthetic when the trunk temperature rose and the palmar temperature stayed down and also in the presence of shock, the palmar temperature fluctuated with the varying conditions of the patient while the forearm and trunk temperatures remained almost unaltered.

These differing reactions to general anaesthesia can be explained by the entirely different nervous control of the cutaneous blood vessels in the two types of skin. Hertzman (1959) states that the vessels in the palmar, plantar and aural skin are innervated by

FIG. II. Oesophageal temperature fell one degree centigrade during this long operation. Palmar temperature showed a rapid rise and fall on induction and recovery, and stayed within one degree of the oesophageal temperature during maintenance. The forearm skin followed a different pattern coinciding with the palmar temperature during part of the recovery phase only. Induction caused a gradual fall in muscle temperature. After turning the patient prone there was a sharp fall which did not return to normal. Two separate inhalations of 20 per cent carbon dioxide caused no changes in temperature.

sympathetic vasoconstrictor fibres only and that vasodilatation is produced by paralysis of these nerves. The vessels of the remaining skin are innervated mainly by cholinergic sympathetic, vasodilator fibres with vasoconstrictor fibres in a definite minority. Paralysis of this nerve supply would lead to an unpredictable resultant blood flow. The physiological control of these vessels is still not completely understood. It is of interest to anaesthetists that the superficial veins of the limbs are controlled by sympathetic, vasoconstrictor fibres only, identical with the palmar and plantar skin vessels even though these veins run beneath areas of skin such as the forearm whose vessels are controlled by the more complicated mechanism.

Further work into the control of the blood vessels of the forearm and trunk skin and the effects of general anaesthesia on them could be of value in the prevention of blood loss during surgery and of the effectiveness of planned hypotension, hypothermia or special techniques used to reduce blood loss during surgery.

TABLE 1

MEAN VALUES OF CHANGES IN PALMAR TEMPERATURES

	No. of cases	*Mean*	*Maximum*	*Minimum*	*No change*
Rise after induction	38	3.24°C	10.5°C		1
Time to reach maximum rise	36	21.4 mins	60 mins	5 mins	
Fall during recovery	39	2.44°C	8.9°C		3
Time to reach maximum fall	34	30.8 mins	120 mins	5 mins	

TABLE 2

MEAN VALUES OF CHANGES IN FOREARM TEMPERATURES

	No. of cases	*Mean*	*Maximum*	*No change*
Rise after induction	33	1.38°C	3.35°C	3
Fall after induction	3			
Time to reach maximum rise	30	24 mins	90 mins	
Fall during recovery	29	1.7°C	6.6°C	4
Rose during recovery	2			
Time to reach maximum fall	25	32.6 mins	90 mins	

TABLE 3

MEAN VALUES OF CHANGES IN TEMPERATURE OF SKIN OF TRUNK

	No. of cases	*Mean*	*Maximum*	
Rise after induction	10	1.61°C	3.1°C	2 fell
Fall during recovery	8	1.23°C	4°C	2 rose

TABLE 4

FOOT TEMPERATURES

Degrees Centigrade

Case number	*32*	*36*	*39*	*41*	*43*
Rise during induction:					
Toe			9.4	3.8	13.6
Heel			4.7	2.5	7.4
Posterior tibial artery			2.65	0.7	4.2
Tendo Achilles	3.8	6	2.7	1.1	2.9
Fall on recovery					
Toe			7	0.8	10.4
Heel			4.3	0.9	4.9

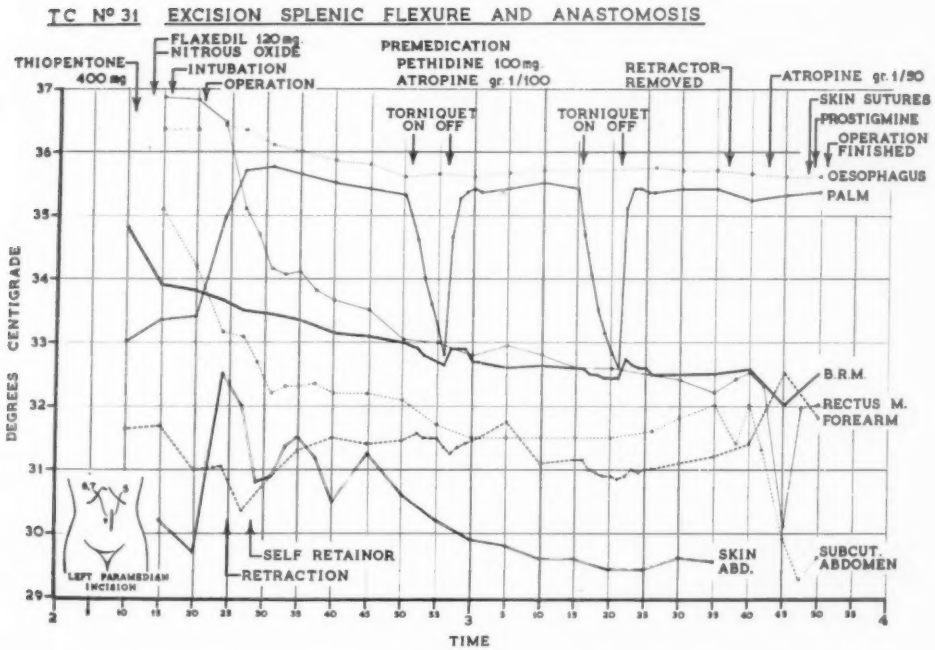


FIG. V. Induction resulted in a sharp rise of palmar temperature which remained within 0.5°C of the oesophageal temperature throughout. The forearm temperature did not follow a similar pattern. The muscle temperature fell gradually throughout. Two periods of arterial occlusion caused sharp drops in palmar temperature and very small drops in forearm skin and muscle temperature indicating a much greater blood flow in the palmar skin than either the forearm skin or muscle. The temperatures recorded near the wound showed the effects of anaesthesia, loss of heat by convection and pressure of retraction.

TABLE 5

MEAN VALUES OF CHANGES IN MUSCLE TEMPERATURES

Degrees Centigrade

	No. of cases	*Mean*	*Maximum*	*No change*
Forearm muscles				
Fall after induction	38	1.86	5.9	2
Rise during recovery	34	0.61	4.3	16
Trunk muscles				
Fall after induction	14	1.25	4.5	0
Rise during recovery	16	1.05	4.3	3

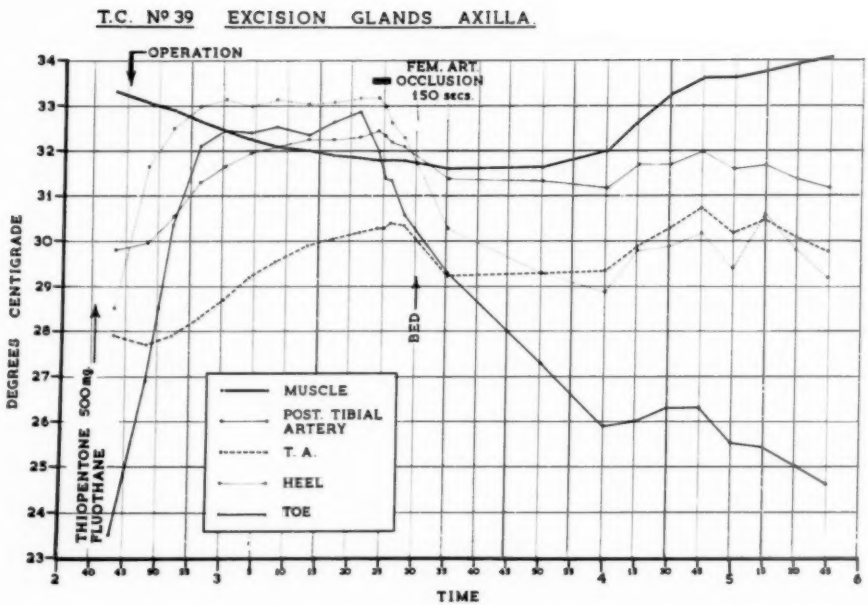


FIG. VI. Induction was followed by a sharp rise in temperature of the plantar skin and there was a fall on recovery. The gastrocnemius muscle temperature fell. Although a bloodless tissue the tendo Achilles absorbed heat by conduction from the adjacent posterior tibial artery carrying the increased blood flow to the plantar skin. The temperature in the tendon increases the significance of the fall in the muscle itself. The muscle temperature returned to normal post-operatively.

TABLE 6

CALF MUSCLE TEMPERATURES

Degrees Centigrade

Case number	*32*	*36*	*39*	*41*	*43*
Fall during induction:					
Superficial gastrocnemius muscle	1.6	0.9	1.7	1.1	1.4
Deep gastrocnemius muscle				0.95	1.0
Rise on recovery:					
Superficial gastrocnemius muscle	0	0.7	2.5	1.1	1.5
Deep gastrocnemius muscle				1.0	0.7

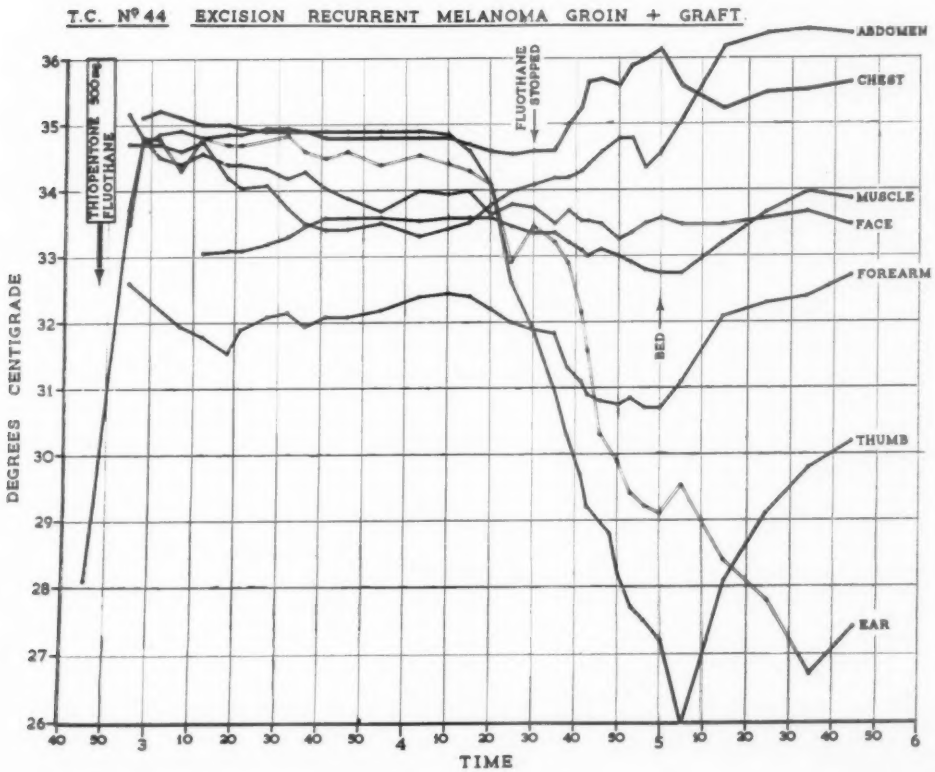


FIG. VII. Induction of anaesthesia resulted in a sharp rise in palmar and aural temperature and steep falls on recovery. The skin of the forearm, face, chest and abdomen reacted quite differently. All temperatures except the ear rose after return to bed.

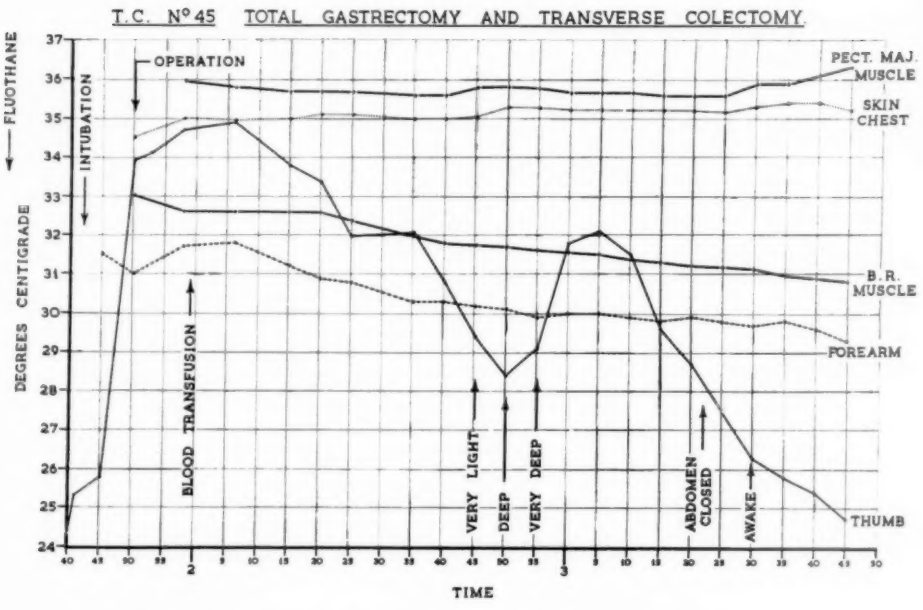


FIG. VIII. This patient was anaesthetized with halothane alone following premedication with atropine. Induction resulted in a steep rise in palmar temperature which was reduced by the effects of considerable blood loss and light anaesthesia. A second temperature rise followed a period of deep anaesthesia. The temperature of forearm skin showed little change throughout. The skin of the chest rose slightly following induction and showed none of the other changes. Forearm muscle temperature fell gradually throughout. The trunk muscle fell slightly each time deep anaesthesia was produced and rose considerably after recovery.

Muscle temperatures

Tables 5 and 6 show that muscle temperatures fell in 50 out of 52 measurements. The falls were greater in the limb muscles than in the trunk muscles but owing to the reduced thermal gradient in the trunk the same fall is more significant. Some of the temperature falls could be ascribed to a reduction in muscle activity, although Pennes (1948) considered that forearm muscle temperature depends on blood flow more than cellular activity. In 37 out of 39 cases the forearm muscle temperature fell below that of the palmar skin. The blood going to the muscle must have been at the same or a higher temperature than that going to the skin of the palm which was exposed to a cooler environment than the forearm muscle. This may not be enough evidence to prove vasoconstriction of muscle blood vessels but it does indicate that there is no vasodilation in muscle vessels during anaesthesia.

Many workers, Shackman, Graber, and Melrose (1952); Prime and Gray (1952); Lee *et alii* (1953); Kitchen *et alii* (1953) have stated that there is an increase in forearm muscle blood flow following the induction of anaesthesia. They drew their conclusions from the use of venous occlusion plethysmography of the forearm and assumed that, as muscle occupied the greater volume of tissue in the instrument, the measured increased occurred mainly in the muscle and not in the skin. Using improved physiological techniques, Roddie, Shepherd and Whelan (1957) and Edholm, Fox and MacPherson (1957) showed that the increase in forearm blood flow during general body heating was confined entirely to the skin, whereas numerous previous workers using the same methods and deductions as Shackman *et alii* (1952) had concluded that there was an increase in forearm muscle blood flow during general body heating. This radical change

by the physiologists demands a re-appraisal of the effects of anaesthetics on muscle blood flow.

The mechanism of control of muscle blood flow is not well understood. Vasodilator and vasoconstrictor fibres have been demonstrated. Mottram (1958), showed that there is little correlation between oxygen utilization or debt and blood flow in muscle, and suggested that there are two types of vessel in muscle tissue, namely metabolic channels and by-pass channels. This is similar to the state of affairs in palmar skin and it is the blood flow through the by-pass channels that is so much increased during general anaesthesia. If there was a similar vasodilatation in muscle during general anaesthesia, then there would be an associated rise of temperature following

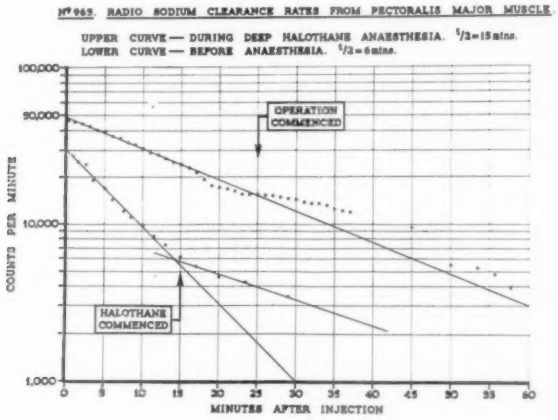


FIG. IX.

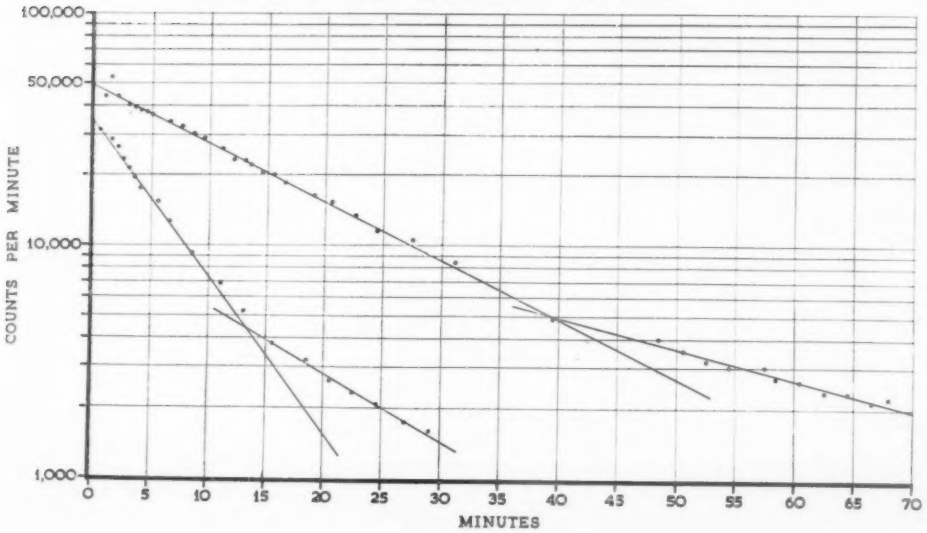


FIG. X.

the induction of anaesthesia. Temperature changes are an indication of the total blood flow through tissues that is the sum of the flow through the metabolic and by-pass channels. Enough evidence is produced here to show that no vasodilatation occurs in muscle during anaesthesia and probably there is a vasoconstriction.

during anaesthesia when compared with the pre-anaesthetic rate. As the clearance time is a function of the metabolic or capillary blood flow, these results indicate that there is a reduction in this fraction of the total blood flow during general anaesthesia. A reduction of cellular activity would not affect this evidence and when considered with the results

TABLE 7

$Na^{22}Cl$ CLEARANCE TIMES FROM MUSCLES DURING ANAESTHESIA

Case	*Muscle*	*Time to reach half count minutes*	
		Before anaesthesia	*During anaesthesia*
1	Brachioradialis	5.0	13.0
2	Deltoid	11.0	11.0
3	Gastrocnemius	8.0	15.5
4	Brachioradialis	9.0	12.5
5	Deltoid	13.3	18.5
6	Pectoralis major	6	15

Radiosodium clearance in muscle

To give further information on the effects of general anaesthesia on the blood flow through muscle tissue an additional six patients were used to determine the clearance times of radiosodium. Veall and Vetter (1958), from various muscles before and after the establishment of moderately deep anaesthesia with halothane, avoiding marked hypotension as far as possible. In each case 0.1 ml physiological $Na^{22}Cl$ (50 uc/ml.) was injected through a very fine needle into the selected muscle and the rate of fall of radioactivity measured with a scintillation probe. After general anaesthesia was induced with halothane vapourized in a circle circuit using voluntary respiration and a moderately deep level of anaesthesia was stabilized, a second dose of 0.1 ml $Na^{22}Cl$ was injected into the same muscle or into the same muscle on the opposite side and the rate of fall of activity measured again. After the necessary corrections for background and recirculation, the results were plotted on semi-logarithmic paper. Two of the resulting curves are reproduced (Figs. IX and X).

The times taken to halve the first counts are shown in Table 7.

In five of the six measurements there was a significant reduction in the clearance rate of the temperature measurements adds further evidence that blood flow through muscle is reduced during anaesthesia.

Postural effects

In one case the brachioradialis and the gastrocnemius muscle temperatures were falling rapidly, as the patient was tilted head upwards; the calf temperature immediately started to rise while the forearm temperature continued to fall. This seemed analogous to the increased forearm muscle blood flow on passive leg raising in the conscious subject. When this experiment was repeated on another subject both the temperatures continued to fall.

On two occasions the patient was turned from the supine to the prone position half an hour after the start of the operation. On each occasion there was a sharp drop of muscle temperature (Fig. II) although the forearm skin temperature was rising slowly, no alteration of the room temperature and minimal changes in the patient's covering having occurred. There was a slight fall in the blood pressure on both occasions. This was considered to be due to a significant reduction in muscle blood flow on turning these patients into the prone position. Another patient was turned from the supine to the lateral position without this extra fall of

muscle temperature. Of the cases in which the muscle temperatures were observed for several hours post-operatively, three remained low and fifteen rose to the pre-operative value or higher. Thus reduction in muscle blood flow during anaesthesia could exactly balance the increase in blood flow which occurs in the palmar, plantar and facial skin and the resultant change which occurs in the remaining skin, resulting in an unchanged total peripheral resistance as far as muscle and skin vessels are concerned. The significance of this finding is that the oxygenation of muscle tissue could be greatly jeopardized during deep general anaesthesia especially in the presence of hypotension due either to deep anaesthesia or haemorrhage. The presence of warm, pink hands with dilated superficial veins along the forearm does not indicate an equally abundant circulation in the muscles of the anaesthetized patient.

Wound temperatures

On 16 occasions, thermocouples were placed within an inch of the surgical wound, mainly laparotomy wounds, the wire or needle electrodes being inserted before the incision was made and fixed in position. Many technical difficulties were encountered, especially in maintaining aseptic conditions, and preventing the dislodgement of the thermocouples during the movement of the tissues being retracted. No definite conclusions could be drawn from the results of these 16 cases but most of them showed the following trends. The skin temperature rose following the induction of anaesthesia, fell during surgery and rose rapidly after wound closure, reaching a higher temperature than the same area before surgery, and a similar area remote from the wound. If any spiritous lotion was applied for skin preparation there was a precipitous fall of temperature which took up to thirty minutes to recover. The subcutaneous tissues showed similar changes to the skin but in a lesser degree. The rectus muscle temperature fell with the induction of anaesthesia and fell even more following incision and retraction of the wound. Owing to the added changes in the environment of the tissues close to the wound, namely room air bathing the deeper layers, retraction of tissues and application of lotions it is not possible to relate temperature changes to changes in blood flow without using more strictly controlled conditions in a larger series of experiments. It was not possible to determine whether the use of antihistamine drugs made any difference to the blood flow near surgical wounds by this method. The effect on the temperature of the tissues exerted by the theatre light used during these cases was investigated on three occasions and it produced no change. Even directing the light directly on a thermocouple itself had little effect.

SUMMARY

By using thermocouples to record changes in the temperature of various tissues, certain deductions in the distribution of peripheral blood flow during anaesthesia have been made. There is near maximal vasodilatation of the vessels in the palmar, plantar and aural skin, which persists for the duration of the anaesthetic provided that shock does not develop. The remaining skin reacts quite differently, the dilatation if present is not maximal.

There is no vasodilatation, and probably a vasoconstriction of muscle blood vessels during anaesthesia. Adoption of the prone position causes a further fall in muscle blood flow. Six measurements of radiosodium clearance rates before and during general anaesthesia gave further evidence of a reduction in muscle blood flow during general anaesthesia.

REFERENCES

EDHOLM, O. G., FOX, R. H., and MACPHERSON, R. K. (1956), *J. Physiol.*, vol. 134, page 612.

FOSTER, A. D., NEUMANN, C., and ROVENSTINE, E. A. (1945), *Anesthesiology*, vol. 6, page 246.

HERTZMAN, A. B. (1959), *Physiol. Rev.*, vol. 39, page 280.

KITCHEN, A. H., SANGER, C., de WARDENER, H. E., and YOUNG, I. M. (1953), *Clin. Sci.*, vol. 12, page 361.

LEE, G. de J., CHURCHILL-DAVIDSON, H. MILES, B. E., de WARDENER, H. E. (1953), *Clin. Sci.*, vol. 12, page 169.

MOTTRAM, R. F. (1958), *J. Physiol.*, vol. 142, page 314.

PENNES, H. H. (1948), *J. Appl. Physiol.*, vol. 1, page 93.

PRIME, F. J., and GRAY, T. C. (1952), *Brit. J. Anaesth.*, vol. 24, page 101.

ROBERTSON, J. D., and SWAN, A. A. B. (1957), *Anaesthesia*, vol. 12, page 182.

RODDIE, I. C., SHEPHERD, J. T., and WHELAN, R. F. (1956), *J. Physiol.*, vol. 134, page 444.

SHACKMAN, R. GRABER, G. I., and MELROSE, D. G. (1952), *Brit. J. Surg.*, vol. 40, page 193.

VEALL, N., and VETTER, H. (1958), "Radioisotope Techniques in Clinical Research and Diagnosis." London, Butterworth, page 371.

THE BEHAVIOUR OF THE SPHINCTER OF ODDI IN HEALTH AND DISEASE*

By T. P. Nash

Sydney

THE sphincter of Oddi is of particular interest to surgeons because of its significant role in post-cholecystectomy syndromes and its aetiological importance in certain types of pancreatitis. Situated at the confluence of biliary and pancreatic streams, it is able to influence function of either system because, contrary to what might be expected, it is a strong sphincter capable of resisting the secretory pressures of both liver and pancreas. This account of sphincter behaviour concerns principally 2 aspects, firstly, the effect of biliary tract disease on the function of the sphincter and secondly, the effect of disordered function which may result in producing post-cholecystectomy pain and recurrent biliary tract disease.

Anatomy

The sphincter of Oddi surrounds the terminal part of the common bile duct and pancreatic duct during their course through the duodenal wall. This intramural course of the ducts is surgically important and the following features should be remembered. The course of the common bile duct through the duodenum is S-shaped and about 1 to 1.5 cms. in length but when the sphincter contracts it becomes a slight curve and the length becomes reduced to about 0.5 cm. The common bile duct is also greatly reduced in calibre as it passes through the duodenal wall and becomes a filamentous canal 2 to 3 mm. in width. The opening in the duodenal muscle through which the ducts pass has a pinchcock action on both ducts and when the duodenum contracts this pinchcock action tends to prevent reflex of duodenal contents. It also limits the size of a calculus that may pass into the duodenum from the common bile duct. The pancreatic duct lies in close relationship to the common bile duct and both are surrounded by the sphincter.

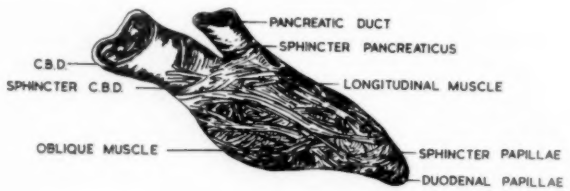


FIG. I.

We recognize 4 main post-cholecystectomy syndromes resulting from residual or recurrent common duct stones, partial obstruction of the common bile duct from acute papillitis or stricture of the ampulla, recurrent pancreatitis and biliary dyskinesia where pain and discomfort are due entirely to sphincteric dysfunction. The clinical features of the first 2 syndromes are so typical and so readily demonstrated by billigrafin examination that the role of the sphincter of Oddi in their causation is often overlooked. In the remaining 2 the sphincteric disturbance is all important, because not only is this disturbance of function the main causative factor, but sphincterotomy is often the only means of relieving the disorder (Fig. I).

The sphincter commences just proximal to where each duct enters the duodenal wall so that a small part of the sphincter is outside

*Received for publication 28th June, 1960.

the duodenum. It ends at the duodenal papillae where the ducts open into the intestine. In length it is about 1 to 1.5 cms. but during contraction is reduced in length to 0.5 cm.

The muscular arrangement of the sphincter is complex but it is so arranged as to provide for the 2 main functions of the sphincter. There is an outer layer, consisting of longitudinal and oblique muscle bundles some of which are arranged around the ducts in a spiral fashion (Fig. I). This is responsible for the shortening and widening of the duct that occurs during evacuation. It also facilitates the discharge of bile and pancreatic juice into the duodenum and overcomes the increasing angulation of the ducts that tends to occur, during digestion, from the increasing intraduodenal pressure.

but several features are of special surgical interest.

Firstly, a part of the sphincter is extramural and surrounds both ducts before they pass through the duodenal wall. It is therefore unlikely to be divided by operative sphincterotomy, and most sphincterotomies must consequently be regarded as anatomically incomplete. In dissected specimens this extramural part of the sphincter extended no more than 2 or 3 mms. along the duct. It consisted of circular and longitudinal layers but both were reduced in size, so that from an anatomical viewpoint it appeared that this portion could be only mildly sphincteric. It was therefore unlikely to influence to any great extent the value of operative sphincterotomy. This view was later confirmed by observing the pressure changes in the common bile duct after sphincterotomy using morphia to cause spasm of this remaining sphincter. A sphincteric action could still be seen, but it was capable only of resisting pressures up to 250 mms. of bile which is little beyond the physiological range.

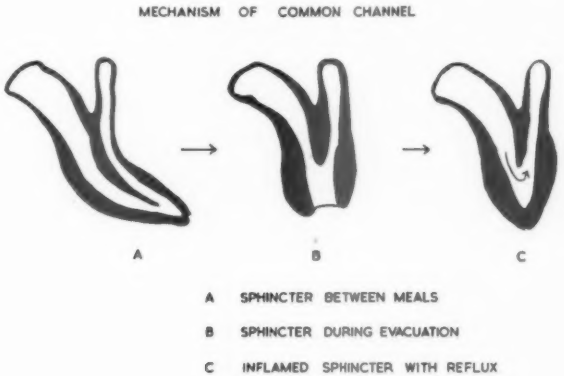


FIG. II.

The inner layer is a more highly developed circular muscle coat. It is the truly sphincteric portion and is a complete layer extending from the papilla to just beyond the duodenal wall. Some circular fibres surround each duct separately but some pass between the ducts in a figure-of-8 manner. The sphincter is usually named in 4 parts, the sphincter choledochus which surrounds the common bile duct, the sphincter pancreaticus which surrounds the pancreatic duct, the sphincter ampullae and the terminal part, the sphincter papillae (Fig. I).

Surgical anatomy

A detailed account of the muscular arrangement of the sphincter is not intended here

Secondly, the sphincter papillae may contract separately from the remainder of the sphincter and this contraction may be sufficient to obstruct bile and pancreatic ducts. In specimens this part of the sphincter may appear small, but when the sphincter contracts it forms a bulky circular layer. Moreover it is the part of the sphincter which is first affected by biliary tract infection.

Next, as the ducts come together in the duodenal wall and are surrounded by the common sphincter, they are separated by a muscular partition (Fig. II). The muscle fibres in this partition are largely longitudinal and spiral giving this partition a tongue-like action and enabling it to retract or elongate as the sphincter contracts or relaxes. This is an important fact in producing a functional common channel between bile and pancreatic systems.

FIG. III. Microphotograph of sphincter showing outer (a) longitudinal, (b) oblique muscle layers, (c) groups of mucous glands between muscle bundles, (d) thick circular inner coat.

Finally, there are groups of mucous glands between the muscle bundles of the sphincter (Fig. III). These glands lie outside and between the muscle layers and their rather long ducts pass through small openings in the bundles to reach the lumen. When the sphincter contracts or becomes spastic the ducts are obstructed. Moreover, when these glands become infected from the gall-bladder or common duct the infection could readily spread amongst the muscle layers of the sphincter. They are important factors in producing acute papillitis and stricture of the ampulla.

PHYSIOLOGY

The sphincter regulates the flow of bile into the duodenum and is responsible for filling the gall-bladder between meals. During fasting the sphincter is tonically contracted and bile is retained against the secretory pressure of the liver. When the pressure in the common duct exceeds 80 mm. the bile flows back into the gall-bladder where it is concentrated. This concentrating power of the gall-bladder acts as a delicate pressure-regulating mechanism within the biliary system and under normal conditions pressure is kept within very narrow limits. This explains why removal of a normal gall-bladder may cause symptoms in certain individuals because, if the sphincter becomes spastic, they are then unable to regulate the pressure within the common duct.

In man the sphincter is mainly under hormonal control. After the ingestion of food there is a short and unimportant nervous flow of bile but, when food reaches the duodenum, the hormone cholecystokinin is released and as a result the gall-bladder contracts and the sphincter relaxes and there is a rich flow of concentrated bile into the duodenum. This action of cholecystokinin on the sphincter is specific and independent of its action on the gall-bladder. It can be readily demonstrated in patients after cholecystectomy with an indwelling T-tube in the common bile duct by the method of Bergh and Layne.

After cholecystectomy the sphincter may lose its tone but it usually returns within the first week. The tone of the sphincter may even increase after cholecystectomy acting, in some measure, as compensation for loss of the gall-bladder and this may cause some enlargement of the common duct. However, the presence of an enlarged or dilated common duct after cholecystectomy should not be regarded as due to increased tone of the sphincter alone, but rather as evidence of persistent disease within the biliary tract.

The sphincter is readily influenced by our emotions and in some individuals this effect may be dramatic and severe causing complete and prolonged spasm. Fear and anxiety are notable examples. This was well seen in a patient who overheard a remark during

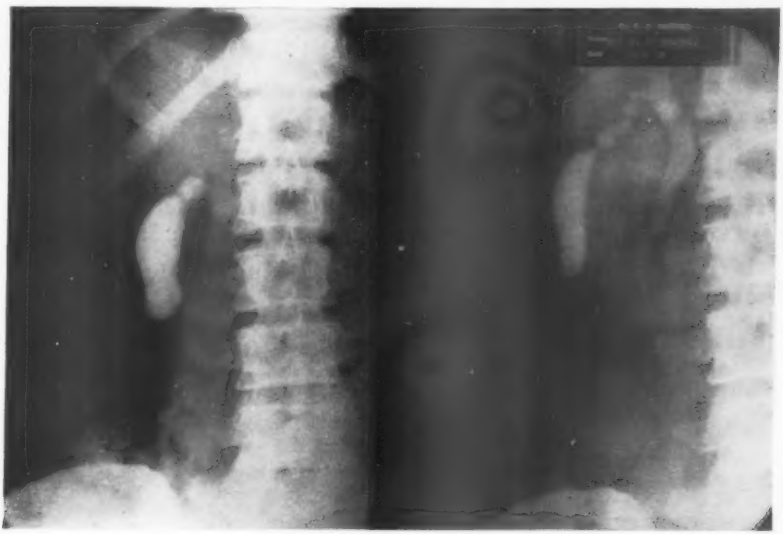
cholangiography that a residual calculus was still in the common duct. The fear of a possible second operation caused complete and persistent spasm.

Although spasm due to emotional factors may occur in a normal biliary system it is unlikely to cause more than transient discomfort because the concentrating power of the gall-bladder by reducing the volume of bile soon lowers the pressure within the common bile duct. However in the presence of biliary tract disease, when the sphincter appears more sensitive and the concentrating power of the gall-bladder usually lost, spasm may then cause definite pain and discomfort. This pain is frequently seen in cholecystectomized patients undergoing some other operation where morphia or omnopon is used as premedication.

marked epigastric pain and vomited. This was subsequently relieved by sphincterotomy.

Actions of drugs on sphincter

The action of drugs on the sphincter is of importance therapeutically but was found to vary sometimes from current and standard beliefs. Morphia and pethidine for example both caused marked spasm. In general the effect of drugs is found to vary from person to person under normal conditions, but when the sphincter is diseased the action of all the drugs causing contraction appears markedly increased. The method used for these investigations was to observe sphincteric changes in a person whose common bile duct had been explored during cholecystectomy and found normal. A suitable time was allowed to elapse for the tone of the sphincter to recover as judged by pressure observations, normal



(a) (b)

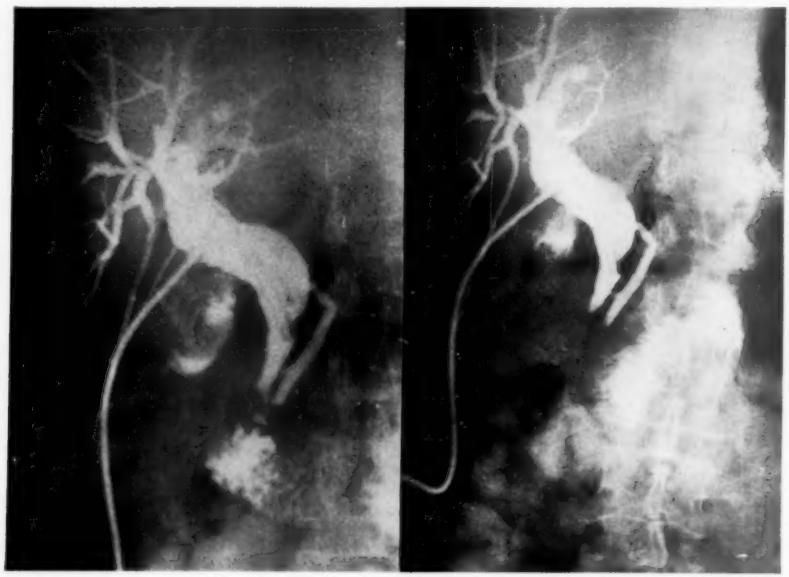
FIG. IV. Cholecystogram of a patient with biliary dyskinesia. (a) A normal cholecystogram. (b) Cholecystogram after a fatty meal. The gall-bladder emptied but the sphincter remained firmly contracted. The patient experienced a mild attack of biliary colic.

An example of a different type of spasm or dysfunction is shown in (Fig. IV). This patient suffered from typical attacks of biliary colic and fatty dyspepsia. A cholecystogram was performed. When the fatty meal was taken the gall-bladder contracted but the sphincter became firmly contracted. At the same time the patient experienced cholangiographic findings and for the patient to recover completely from the operation. The investigations were performed in the radiology department with a screen and image intensifier available. Using this method on suitable patients the following results were obtained. Morphia and pethidine were constant in causing spasm. It was not relieved

by atropine. This spasm may last several hours and varies to some extent in different patients. When the sphincter was diseased or inflamed this spastic effect was greatly enhanced and as little as morphia gr. 1/12 caused severe spasm. It was of interest to observe that, with very small amounts of morphia, spasm of the sphincter papillae occurred first. Several other morphine derivatives were used and all were found to be spastic in effect. The relaxing effect of probanthine, merbentyl and felicar could not be demonstrated when spasm was induced by morphia. The only known substances which relax the sphincter are amyl nitrite, nitroglycerine, magnesium sulphate and egg yolk. The effect of amyl nitrite was rapid in the effect and decreased to some extent the spastic effect of morphia.

and sub-acute pancreatitis associated with biliary tract disease. In fact it is the most important cause of pancreatitis (Fig. Va and b).

This role of biliary reflux as a causative factor in pancreatitis has intrigued clinicians for over fifty years since Opii first described a case of reflux pancreatitis resulting from a stone impacted at the ampulla of Vater. The objection to the biliary reflux theory has always been an anatomical one, that the common channel is present in less than 30 per cent. of cases. This is the usual finding when specimens are examined but fails to take into account that the sphincter in life is not a fixed structure but an actively mobile organ capable of readily changing in character. Doubillet and Mulholland have demonstrated



(a) (b)

FIG. V. Cholangiograms showing the effect of morphia in producing spasm of the sphincter; (a) before, (b) ten minutes after injection of morphia.

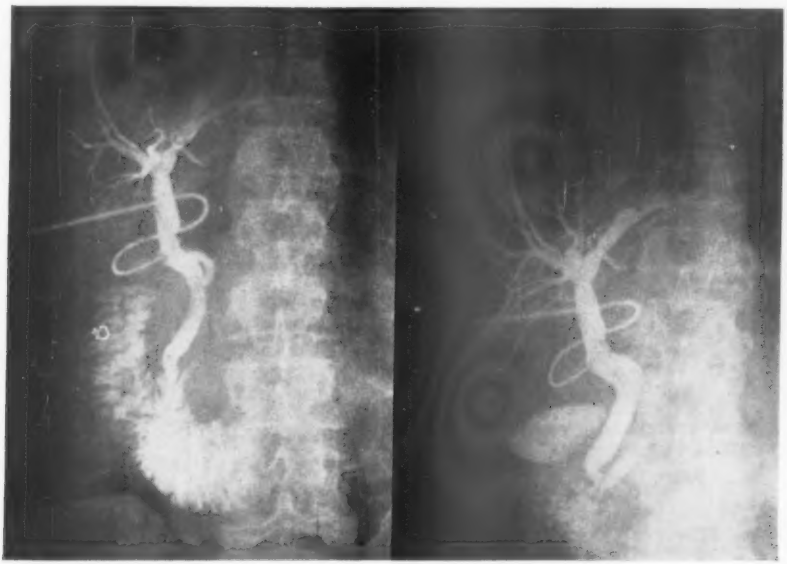
THE COMMON CHANNEL AND PANCREATITIS

Sphincter spasm due to infection within the common bile duct or gall-bladder is, without doubt, the most important feature of abnormal sphincter behaviour. Not only is this spastic sphincter a most important factor in causing biliary colic but it is responsible for the development of all or almost all acute

beyond doubt that a functional common channel does exist. They were able to demonstrate such a channel in 314 out of 317 cases examined. Their method was to induce spasm of the sphincter papillae by application of a little acid locally through the duodenal wall and at the same time perform an operative cholangiogram.

The common channel is produced as a result of 2 factors. Firstly, there is persistent spasm of the sphincter papillae as a result of infection and secondly, there is retraction of the muscular tongue-like partition between the ducts which occurs when the sphincter normally widens and retracts during evacuation. When this occurs bile or, in the case of pancreatitis, probably infected bile is able to reflux readily along the pancreatic duct. It has been stated, from observations in dogs, that the pressure of pancreatic juice in the pancreatic ducts exceeds the pressure of bile in the common bile duct and that biliary reflux along the pancreatic duct would consequently be unlikely. In humans, however, who have biliary tract infections, biliary reflux along pancreatic duct was quite readily demonstrated under low pressures. In a group of 11 patients with pancreatitis associated with gross biliary tract disease and in whom T-tube drainage was instituted as part of the definitive treatment of their pancreatitis, marked reflux occurred in 8 cases (Fig. VIa and b). Biliary reflux under low pressures has been similarly seen during routine cholangiography. One other reason for this reflux when a common channel exists is that the pressure in the common bile duct may exceed normal ranges due either to previous cholecystectomy or absence of the concentrating power of gall-bladder from infection. In other words the loss of the chief pressure regulator of the biliary system may result in increases in common bile duct pressure to exceed that within the pancreatic duct.

The evidence that biliary reflux is in fact a causative factor in these cases of pancreatitis associated with biliary disease may be summarized.



(a) (b)

FIG. VI. Post-operative cholangiogram of a patient who was operated on for acute pancreatitis with associated biliary tract disease. (a) There is a little reflux along the pancreatic duct but most of the dye enters the duodenum. (b) A few minutes later there is more marked reflux along the dilated pancreatic duct.

1. Most cases of recurrent pancreatitis will resolve after removal of the associated biliary tract disease.

2. Of the cases persisting after normal biliary surgery many are resolved by interrupting the common channel by sphincterotomy.

3. The finding of bile and pancreatic juice in the retroperitoneal tissues around the pancreas in cases of pancreatitis, which could only be explained by rupture of a pancreatic ductule.
4. Demonstration of a common channel in 8 of 11 cases of pancreatitis by normal cholangiography.
5. Marked improvement in these cases of acute pancreatitis by T-tube drainage of the common bile duct.

THE SPHINCTER AND BILIARY PAIN

It is commonly stated that biliary colic is the result of spasm of the common bile duct or gall-bladder around a stone, or active muscle contraction of either gall-bladder or common bile duct aimed at expelling the stone. Yet the contracting power of the gall-bladder is weak and is lost in the presence of infection and the common bile duct contains little or no muscle, certainly insufficient to cause spasm. Two methods were used in this

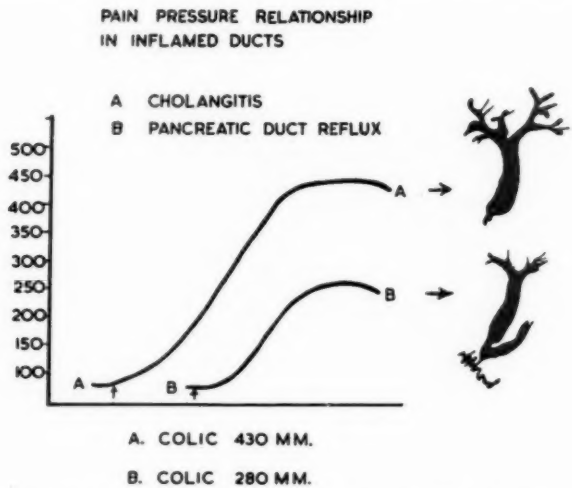


FIG. VII. Pain-pressure relationships in inflamed ducts. Biliary colic occurred at a pressure of 430 mms. of bile in a patient with cholangitis. When the pancreatic duct was distended biliary colic occurred at a pressure of 280 mms.

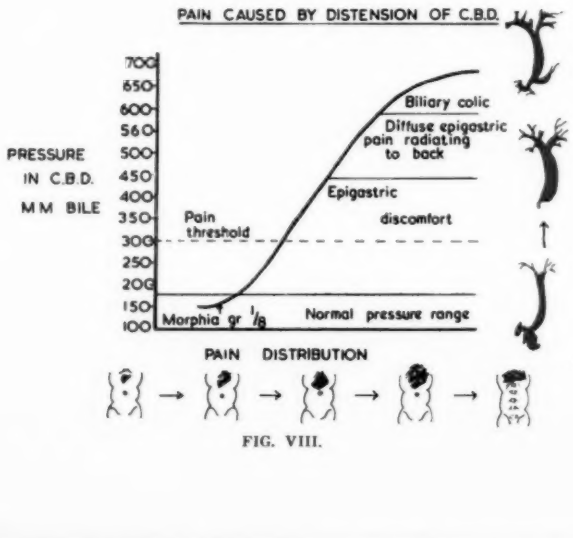


FIG. VIII.

investigation of biliary pain. One was to correlate the patients' symptoms with the operative findings when acute surgery was performed for cholecystitis or pancreatitis. The second method was to attempt to reproduce the same pain in these patients in which it was found necessary at operation to drain the common bile duct with a T-tube. The results of these observations left little doubt that the principal factor in biliary pain is distension of either an inflamed pancreatic or common bile duct resulting, initially, from spasm of the sphincter of Oddi. It was found that distension of an inflamed pancreatic duct produced more severe pain than distension of the common bile duct at the same pressure, but that the character of the pain resulting from distension of either duct was approximately the same. Operative findings showed quite clearly that patients with typical biliary colic may have either an attack of reflux pancreatitis, acute cholecystitis or acute papillitis. It is interesting to reflect that many patients with typical biliary colic and treated as such by an injection of morphia or pethidine have in fact an attack of pancreatitis.

The pain-pressure relationships in the common bile and pancreatic duct are set out in Figs. VII and VIII together with the pain distribution and the cholangiographic findings. These were performed by inducing sphincter spasm with a small injection of morphia. Pain was readily produced by distending a normal common bile duct but the pressure necessary to produce biliary colic was found greatly in excess of the physiological range. When an inflamed common bile duct was distended however, pain amounting to biliary colic was readily produced by pressures well within the physiological range and when reflux along the pancreatic duct occurred, the pressure necessary to cause biliary colic was even less. It will be noticed that with distension of ducts at low pressures the patient experiences an epigastric discomfort which they describe as being identical with the flatulent dyspepsia many experience with their gall-bladder disease.

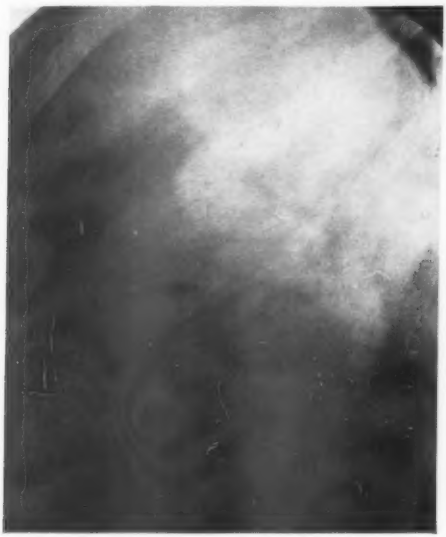


FIG. IX. A typical finding in a patient with strictures of the ampulla of Vater on billigrafin examination.

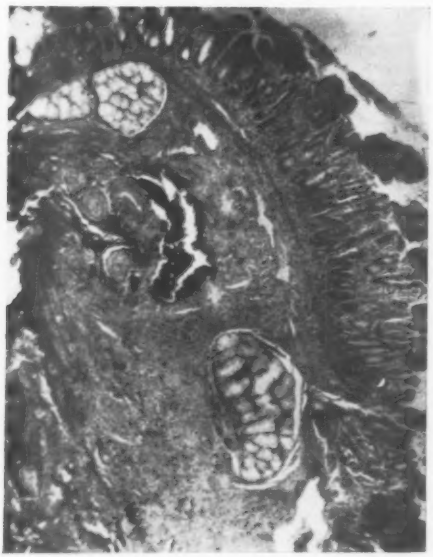


FIG. X. Microphotograph of a biopsy specimen taken from a patient whose billigrafin is shown in Fig. IX while sphincterotomy was being performed. There is almost complete destruction of the muscle of the sphincter.

MECHANISM OF ORGANIC DISEASE OF SPHINCTER

After cholecystectomy or choledocotomy most biliary tract infection is relieved and the sphincter returns to normal. In some cases however, infection may persist in the intramural portion of the common bile duct. The reasons for this are that the duct here is very narrow, the mucosal lining is redundant and thrown into folds which easily occlude the lumen when inflamed, the presence of numerous mucous glands which are readily infected and the sphincter which may be spastic. This smouldering residual infection tends in time to produce varying degrees of biliary obstruction from oedema and fibrosis of the sphincter and the patient may develop attacks of pain and jaundice (Fig. XII). Jaundice tends to occur from either an exacerbation of the existing infection producing acute papillitis, or from fibrotic stenosis. As the common bile duct obstruction increases, recurrent stones may form. A billigrafin examination usually reveals a dilated common bile duct with delayed emptying. Biliary cirrhosis occurs when partial obstruction has been longstanding. Fig. VII shows the billigrafin of a patient with common bile duct obstruction due to chronic papillitis. The papillitis has progressed almost to the stage of complete fibrous stricture seen in Fig. VIII. There is almost complete destruction in the sphincter muscle. When any degree of obstruction to the common bile duct results from either papillitis or stricture, sphincterotomy becomes essential. Damage of the sphincteric muscle resulting in the papillitis or stricture may occur from other causes than residual infection, for example irreversible damage may result from an impacted intramural calculus or from the injudicious use of sounds at choledocotomy (Fig. IX). It is usual practice to perform sphincterotomy whenever an impacted calculus is found or undue difficulty experienced with dilating the ampulla of Vater. One point which should be stressed is that the development of obstruction from fibrous stricture is a very gradual process. One patient remained well for 8 years following cholecystectomy before her stricture of the ampulla became evident and produced attacks of pain and jaundice. This should perhaps make for more careful examination of the sphincter in every case of cholecystectomy and should certainly call for a more painstaking and careful approach when dilating the sphincter during choledocotomy.

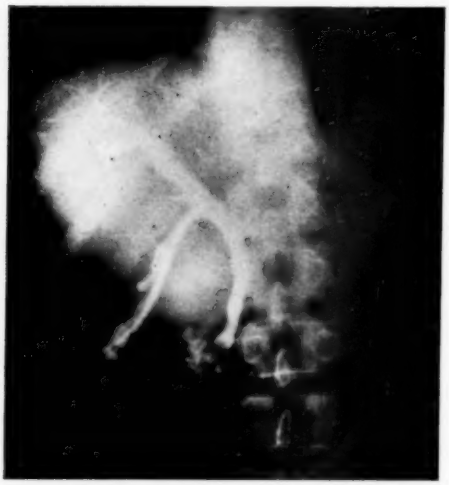


FIG. XI. Post-operative cholangiogram of patient with acute papillitis. The crescent-shaped lower end of the common bile duct here simulates a retained intramural calculus. Sphincterotomy was later done on this patient.

MECHANISM OF STRICTURE

Cholecystitis — Cholangitis

Marked Persistent — Cholangitis of intramural C.B.D.

because:
(a) Duct narrow
(b) Redundant mucosa
(c) Spastic sphincter
(d) Mucus glands infected

Acute papillitis (from glandular infection) — Cholecystectomy usually relieves

Stricture of ampulla from muscle fibrosis

FIG. XII.

SUMMARY

It has been the purpose of this paper to emphasize the importance of the sphincter of Oddi as a factor in the causation of many aspects of biliary and pancreatic disease, in particular its role in the mechanism of biliary pain and its importance in the causation of those cases of pancreatitis associated with biliary tract disease. This investigation has shown how sphincteric dysfunction in the absence of any notable biliary tract infection may cause attacks of epigastric discomfort and confirms the view that biliary dyskinesia is a clinical entity requiring on occasions sphincterotomy. It has shown how persistent infection in the common bile duct even after cholecystectomy or choledocotomy may result in biliary obstruction from inflammation and fibrosis of the sphincter and underlines the importance of careful examination of this region in all cases during biliary tract surgery and the necessity for careful manipulation whenever the sphincter of Oddi requires dilation.

ACKNOWLEDGEMENT

I would like to express gratitude to the Staffs of the Pathology, Radiology and Photography departments of St. Vincent's Hospital for their invaluable help in performing this clinical research and also to my surgical colleagues for making available many of the patients with biliary tract disease for investigation.

REFERENCES

BERGH, G. S. and LAYNE, J. A. (1940), *Aust. J. Physiol.*, vol. 128, page 690.

BOYDEN, E. A. (1937), *Surgery*, vol. 1, page 25.

—— (1957), *Surg. Gynec. Obstet.*, vol. 104, page 641.

COLE, W. H. and GROVE, W. J. (1952), *Ann. Surg.*, vol. 136, page 73.

DOUBILET, H. (1956), *Surg. Clin. N. Amer.*, vol. 35, page 855.

ODDI, R. (1887), *Arch. ital Biol.*, vol. 8, page 317.

SCHREIBER, H. (1944), *Arch. klin. Surg.*, vol. 205, page 211.

CONSERVATIVE SURGERY IN TUBAL ECTOPIC GESTATION*

By S. DE C. BARCLAY

Greymouth, New Zealand

FROM time to time many general surgeons and all gynecologists have to deal with the problem of tubal pregnancy. Until recently salpingectomy has been virtually the only operation in use. For some five years I have been practising conservative surgery for this condition and the results have been so satisfactory that it seems proper to urge this upon others. Conservative tubal surgery so far seems applicable alike to unruptured gestations, tubal abortions and tubal ruptures. My experience is not great but is sufficient to indicate the probability that conservative methods can safely be used in almost every case.

It is beyond my competence to discuss the causes of tubal pregnancy, the liability to recurrence, the functional state of the tube or tubes or anything beyond the simple facts. If both tubes are excised, pregnancy is impossible. If an affected tube can be retained and subsequently shown to be patent without added risk to the patient or any other added difficulty, surely it ought to be retained. Its functional efficiency may be below normal, there may be risk of another tubal pregnancy, but these are unimportant beside the fact that such a tube may function and may be the means of the woman bearing a child.

If the gain were only at the price of another operation or of a longer, bloodier or more upsetting procedure than salpingectomy it would be proper to belittle the value of mere tubal patency and right to go deeply into the intricacies of tubal functions and physiology before recommending conservative surgery. But where the gain, as here, is without cost to the patient it would seem sufficient to accept the value of these operations as measured by the simpler standard of tubal patency.

*Received for publication 7th September, 1960.

Accepting for the moment the desirability of conservative surgery for tubal pregnancy, it becomes the more important to make the diagnosis early and operate promptly. Repeated oozing into the pelvic cavity for days or weeks produces a pudding-like conglomerate of partly organized clot which when cleared away, sometimes tediously, may leave oozing surfaces and reveal a very friable tube remnant. The price of late diagnosis may still sometimes have to be excision of the tube.

The aim of conservative surgery is to evacuate the conception and its products. If one recalls the mode of implantation of an ovum it is clear that the whole circumference of the tubal lumen is never destroyed. A part is, the rest is simply stretched by the enlarging ovum. A strip therefore, and usually an ample one, remains to reform a tube. Two examples are relevant. O'Regan (1940) reported, following earlier authors, treatment of acute salpingitis by slitting open the tubes from end to end. He demonstrated reformation of a lumen and tubal patency. Denis Browne (1950) demonstrated the soundness of the basic concept by forming a urethral tube by burying strips of perineal skin. Strictures do not occur in these tubes.

The methods of conservative surgery are:

(1) Expression

The tube is milked into the abdomen by gentle finger manipulations and if it seems certain that all conception products have been expressed, gentle pressure on the site of implantation is maintained for four minutes by the clock. In some instances where the pregnancy is in the ampulla the fimbriated ostium and ampulla may be packed widely open with "gelfoam." I think this is an advantage when the rawed surface pouts into the peritoneal cavity.

This method is suitable for tubal abortions or unaborted unruptured early pregnancies.

If bleeding continues and distends the site of implantation, if incomplete expression is feared, or if there has been tubal rupture, evacuation is required.

(2) Evacuation

An incision is made into the lumen of the tube at the thinnest point. If rupture has occurred there is already an opening. The edges of the opening are very gently held back and all blood clot and foetal debris very gently sucked, squeezed and eased out. If simple pressure does not stop the bleeding "gelfoam" or "oxycel" is used within the lumen and fine point diathermy used for subserous or muscular bleeders. The incision can be left open or the peritoneum and outer tube wall very lightly and neatly closed over the cavity. I have usually closed the opening by suture.

(3) Resection and repair

This is sometimes called for when there has been a gross rupture of some duration and there is a firmly adherent partly-organised mass of blood clot which, peeled away, leaves tissues too friable and disorganized to do anything with, and in some juxta-uterine lesions. Where there is not an adherent mess of clot or fibrin and where the tubal tissues are reasonably good the affected segment is narrowly excised and end-to-end anastomosis or implant into the uterus done. But if conditions are in any way bad the affected segment is narrowly excised and each end of the tube proper tied with fine black thread, the ends left 1" long, covered with peritoneum, and the abdomen closed. Repair is then done later.

These are simple methods. Bleeding can always be controlled by fingers whilst the tube is mobilized. Often the assistant can control bleeding by clamping tube and ovary between his fingers so they lie displayed in his open palm. If a clamp is used a large Negus tonsil artery forcep is a good weapon, better than arterial clamps. It is often better held in the assistant's hand and not closed fully. For suturing, 5/0 silk on a straight arterial needle is ideal, a curved ophthalmic needle will do nearly as well. Thread, 100 linen, on a fine curved intestinal needle, will serve. Fine splinter forceps are useful for pinpoint coagulation of bleeders. Haemostasis must be as complete as in fenestration or full thickness skin grafting. Diathermy should never be used within the tube lumen proper, only in the wall or subserous layers. The current should be reduced.

It is important to realize that the instruments, needles, sutures and methods used in hysterectomy or cholecystectomy have no value or place in tubal surgery. The most delicate instruments, the finest sutures and needles alone approach the standards of delicacy of touch and handling and precision of technique required. Yet the surgery is not difficult. Once bleeding is controlled the operator may proceed with the deliberateness required to do his neatest work. He may be better seated. If his sight is failing or his hand becoming rough let him direct his assistant. It is a fortunate thing that the surgery of this condition often falls to the younger men who are best equipped by nature for really fine precision work.

It is pertinent to say that what has proved safe and uneventful in this small series might well be bloodily disastrous if the idea were seized on and applied by surgeons who have lower standards either of equipment or haemostasis.

RESULTS

(1) I have treated 11 patients by expression (3) or evacuation (8). In none was there any anxiety during operation, any difficulty in stopping bleeding, or any complications of any sort afterwards. In 10 of these patients patency of the treated tube was demonstrated, at the first attempt, by hysterosalpingography within three months of operation. In one patient the examination attempted three weeks after she left hospital, was terminated when the uterine cavity showed as a fine saucerlike shadow. The filling defect was a normal pregnancy which proceeded to term.

Of the 11 patients three have since had children, none has had a further ectopic pregnancy, one aborted at four months.

Two patients of the 11 have previously had a tube excised for ectopic gestation. One of

these is the patient who aborted at four months, the other has not become pregnant again.

(2) During the five years since the first conservative operation I have twice performed salpingectomy. One of these patients I would now be prepared to treat conservatively. I am doubtful about the other.

(3) Two patients have had other conservative operations. One had an implant of the distal tube into the uterus for a pregnancy that was partly interstitial. Patency was demonstrated. I do not know the late result. One patient had resection and end-to-end anastomosis done. This tube was not patent on testing later.

FIG. 1. Hystero-salpingogram showing the tube after two conservative operations for mid-tubal pregnancy (Case 1).

CASE REPORTS

Case 1

In December, 1956, Mrs. X. had her left tube excised for an ectopic pregnancy. In January, 1958, she again developed a tubal pregnancy. I opened the abdomen, cleaned out the blood and clots, and squeezed the pregnancy mass out of the abdominal ostium. There seemed a small amount remaining in the tubal wall so I opened the tube by diathermy and removed the fragments, then after stopping bleeding by pressure closed the opening and the abdomen. A hystero-salpingogram three months later showed a patent tube with a good peritoneal spill. Her doctor tells me she has not yet conceived, but the point is that she may.

Case 2

Mrs. Y. in 1954 had a right tubal abortion. At operation the pregnancy was expressed and the tube irrigated with saline. The left tube was found distended with bloody fluid and buried in old pelvic adhesions from peritonitis at the age of 3, so salpingostomy was done. The result was that the outer end of the left tube again sealed off but salpingograms showed some peritoneal spill on the right side.

In 1956 she was investigated by Professor W. C. W. Nixon of the Obstetric Unit, University College Hospital, London, who wrote to me "the left tube is sealed with a fairly large hydrosalpinx. The right tube . . . is definitely patent . . " This lady may yet conceive. If it became necessary she has a left tube which might be opened up. But note that her paient tube is the one from which the ectopic pregnancy was removed.

Case 3

Mrs. Z., recently married, was found at operation to have a leaking 1 cm. diameter pregnancy in the mid-portion of the left tube. This was expressed and in order to make quite sure the tube was incised, the implantation site inspected and bleeding stopped by pressure and the tube closed. The right tube appeared normal. She has since had a normal pregnancy.

These are examples of conservative surgery for tubal pregnancy. One patient had only one tube, one had one tube pregnant and one blocked, one had an apparently normal second tube. It seems possible for all of them to have children. If they have further ectopic pregnancies these should again be able to be treated without sacrifice of a tube. If they should for any reason develop tubal blockage something may still be able to be done.

SUMMARY

Management of tubal pregnancy by expression or evacuation, preserving the Fallopian tube is advocated, the method in use is described and results to date given. In 11 cases these methods have been applied without incident and in ten tubal patency has been demonstrated (pregnancy prevented testing in the eleventh).

CONCLUSIONS

Since the method appears free from added risk and of frequent applicability, conservative surgery should be practised in the management of tubal pregnancies. Judgement, and criticism of the functional status of the retained tube, should be withheld until a very large number of these operations have been done and the late results carefully studied. It is meantime sufficient, and in accord with the best principles of surgery, to urge the general adoption of conservative surgery for these patients and await the verdict of the years.

ADDENDUM

Since this paper was written, three more patients have been treated. One of these was Case 1, who in January, 1960, had another pregnancy in the mid portion of the right, and only, tube dealt with as before. A hystero-salpingogram subsequently showed a good lumen throughout, but absence of spill. This occlusion may yet respond to treatment.

REFERENCES

BROWNE, D. (1950), in "Techniques in British Surgery." London, W. B. Saunders.

O'REGAN, R. (1960), *N.Z. Med. J.*, vol. 39, page 93.

ISLET CELL TUMOUR OF THE PANCREAS*

A CASE REPORT

By R. W. Beal†

Clinical Research Unit, Royal Prince Alfred Hospital, Sydney

ISLET cell tumours of the pancreas are medical rarities but must be borne in mind in the differential diagnosis of any patient with hypoglycaemia, which is not a medical rarity, since it may occur in a number of physiological and pathological conditions.

Langerhans in 1869, while still a medical student, described the islet cells of the pancreas which now bear his name. Nicholls in 1902 was the first to describe an adenoma of the cells of Langerhans, but the clinical aspects of the disease remained unelucidated until the physiology of the pancreatic secretion was described by Banting and Best in 1922. Their co-workers Campbell and Fletcher in the following year described the effects of hypoglycaemia induced by excess injected insulin and it remained for Seale Harris (1924) to record his observation that, when he first saw the insulin reaction in a diabetic patient, he realized that he had seen non-diabetic patients who had complained of the same symptoms. He described five cases of hyperinsulinism in the same report. Wilder (1927) reported a case of hyperinsulinism in a forty-year-old physician who at autopsy was found to have a carcinoma of the pancreatic islet cells. Howland *et alii* (1929) from Toronto reported the first successful removal of an islet cell tumour by Roscoe Graham.

In the ensuing thirty years, several reviews of the literature have appeared. Whipple and Frantz (1935) were the first to publish a series of any magnitude and Crain and Thorn by 1949 were able to review 258 published cases and the progress in diagnosis and treatment that had been made up to that time. In 1950, Howard *et alii* were able to find almost 400 reported cases, while Aird (1957) set the number of published cases between 400 and 500.

Greenaway *et alii* (1946) published the first cases reported in the Australian literature. Brown (1956) described 4 cases of his own and 4 cases of his colleagues, which had come to surgery. The only other case which can be found was that of Maxwell (1949), who described a patient with classical symptoms, in whom no adenoma could be found at laparotomy, following which the symptoms persisted.

A case is reported which emphasizes certain of the diagnostic features of pancreatic islet cell tumours.

Case History

L.C., a 45 year-old male, worked in the carding room of a cotton mill. There was no family history of hyperinsulinism or diabetes. Apart from an appendicectomy in 1931, there was no history of past ill-health.

In 1956 he began to experience attacks of weakness, sweating and trembling, which began most often in mid-afternoon, following the completion of his daily work. These sensations persisted on occasions for some months and were usually relieved by rest and food taken on arriving home. Medical advice was not sought until August, 1956, when, on his way home from work, he collapsed and remained unconscious for at least an hour. A physician later saw the patient in consultation and following a random (3 p.m.) blood sugar estimation of 70 mgm. per 100 ml., suggested further investigation, having made the provisional diagnosis of hyperinsulinism. However, as symptomatic relief could be obtained by rest and the taking of food, he did not come to hospital and had no further difficulties for nearly three years.

In June, 1959, he had a moderately severe attack of a prevalent "viral" infection which left him

*Received for publication 22nd July, 1960.

†Supported in part by the National Health and Medical Research Council of Australia.

anorexic and it was during the period of recuperation that his wife came into his room in the afternoon and found him unconscious. His local doctor, on learning of his previous history, gave him intravenous glucose and he woke immediately. He stated afterwards that during this episode he was aware of what was going on around him but was unable to do or say anything.

A glucose tolerance test was performed on 19th June, 1959 (Fig. I) and it was reported upon as showing no significant abnormality. As his symptoms were increasing in severity, admission for investigation was considered imperative and he entered the Clinical Research Unit of the Royal Prince Alfred Hospital on 16th July.

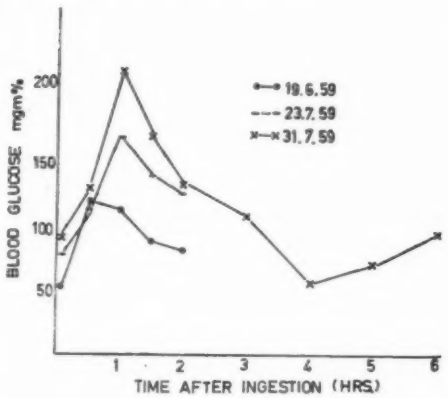


FIG. I.

On examination he appeared to be in good health and no abnormality could be detected in any system. Clinically, there was nothing to suggest liver disease, nor malfunction of any of the endocrine glands. A full blood count, liver function tests, serum electrolytes and urinalysis were all reported as normal. The blood urea was 43 mgm. per 100 ml. and the serum amylase 260 units per 100 ml. of glucose. A standard glucose tolerance test on 23rd July showed no abnormality (Fig. I) and the fasting blood sugar was not less than 90 mgm. on three occasions.

From 6 p.m. on the night of 26th July, fasting was commenced and the next day the patient was encouraged to move about and exercise as much as possible. By midday he was experiencing his customary symptom pattern of weakness, giddiness and profuse sweating, all of which were relieved by sitting down for a short time. While these symptoms were present, twenty-two hours after the commencement of the fast, a venous blood sample was taken and was reported to contain 43 mgm. of glucose per 100 ml., and accordingly he was fed.

A six hour glucose tolerance test was carried out on 31st July (Fig. I) using a standard 50 gm. load and, between the third and fourth hours after the ingestion of the glucose, he began to experience his usual symptoms of hypoglycaemia. The blood sugar level at the fourth hour was 60 mgm. per 100 ml.

He was seen in consultation by Mr. J. S. MacMahon who felt that although the history was not typical, laparotomy was indicated. Before operation it was learned that the blood sugar level of 43 mgm. per 100 ml. reported after fasting on 27th July was in fact 53 mgm. per 100 ml. and further fasting was considered necessary to satisfy Whipple's diagnostic triad (Whipple, 1938). Fasting was commenced on the night of 9th August at 10 p.m. and frequent blood sugar estimations were performed on samples of venous blood. The results of these are set out in Table 1. On the morning of 11th August, the patient awoke complaining of his usual hypoglycaemic symptoms and behaved in a most irrational way so that he had to be restrained. At 1.30 p.m. that day, thirty-nine and a half hours after the commencement of the fast, he went into coma, from which he was roused immediately by an intravenous injection of 50 per cent. glucose. At this stage the blood sugar level had fallen to 39 mgm. per 100 ml.

TABLE 1

Duration of fast (hrs.)	*Blood sugar (mg./100 ml.)*
12	97
14	79
18	61
24	17
36	48
37	48
38	53
39½	39

On 13th August, 1959, under general anaesthesia, Mr. MacMahon performed a laparotomy, the abdomen being opened through a transverse supraumbilical incision. The stomach and pancreas were then mobilized, but after the pancreas had been turned over and palpated thoroughly along its whole length, no tumour could be found and only when the tip of the tail was mobilized from the splenic hilum retroperitoneally was the tumour located. It was approximately 1½ cms. in diameter, reddish-brown in colour and would not shell out. The tip of the tail of the pancreas together with the tumour was excised and the abdomen closed. Intravenous glucose (5 per cent.) was administered throughout the operation and for forty-eight hours subsequently. A transfusion of 500 ml. of whole blood was given in the operating theatre. He remained on intragastric suction for twenty-four hours. Frequent blood sugar estimations were done post-operatively and ranged from 230 mgm. per 100 ml. while still on intravenous glucose, to 106 mgm. per 100 ml.

five days after operation. After fasting for twenty-four hours, the blood sugar level on 28th August was 79 mgm. per 100 ml. and he had no symptoms of hypoglycaemia.

Dr. V. J. McGovern gave the following report on the two specimens sent for histopathological examination. "Macroscopic: (A) The specimen consists of a nodule measuring 2 x 1½ x 1½ cms. It has a creamy yellow appearance with dark patches of haemorrhage. (B) The specimen consists of a flat nodule of tissue. Microscopic: (A) The lesion is a nesidiocytoma. (B) The specimen consists of normal pancreatic tissue."

The patient was discharged on 29th August to the care of his referring physician. He appears to have made an excellent recovery and is devoid of his symptoms, even after moderate exertion.

Discussion

Pathology

When Whipple and Frantz described the first large series of islet cell tumours of the pancreas in 1935 it was considered that the tumours were more common in the body and the tail and this view is perpetuated even in recent textbooks (Boyd, 1958). Aird (1957) however, was careful to point out that while the first 200 described were more common in the body and tail of the pancreas, the distribution of the 400 or more now described is evenly throughout the head, body and tail. The reason proffered by Aird is that "the more modest and retiring tumours, lying in obscurity behind the head of the pancreas, or in the groove between the pancreas and duodenum, were missed in the early days." Be this fact or be this fancy, most authors who have reviewed the literature now agree that the distribution is fairly uniform. Multiple adenomata are reported in 10 to 12 per cent. of cases while in 2 per cent. there are diffuse adenomata of other endocrine glands as well (Crain and Thorn, 1949). It is of some interest to note that the first case described by Greenaway *et alii* had a large cystic tumour removed from the body of the pancreas in 1942 and in 1944 following a recurrence of symptoms, had a smaller tumour removed from the tip of the tail of the pancreas.

Islet cell tumours are said to be benign in 75 per cent. of cases, malignant with metastases in 5 to 10 per cent., while the remaining 15 to 20 per cent. are reported on by the examining pathologist as showing certain of the characteristics of malignancy, such as invasiveness and lack of a well-defined capsule, but which show no tendency to metastasise and are usually regarded, in so far as their behaviour is concerned, as benign (Aird, 1957; Crain and Thorn, 1949). The tumour removed from the patient reported here was haemorrhagic and multilobular, lacking a well-defined capsule but it was regarded as being essentially benign.

Clinical features

The strongest impression left after a survey of the symptomatology of the various large series reported is of the wide range and diversity of the clinical features, there being seldom a case which fits the classical description in every respect. It is well known that patients may be seen at neurological clinics, endocrine clinics and psychiatric clinics before the correct diagnosis of islet cell tumour of the pancreas is made. Renold and Thorn have observed that once a syndrome pattern develops in a patient with an islet cell tumour, it seldom changes and remains constant in that patient no matter how far removed from the textbook description the particular symptom-complex may be. This concept is well borne out by the patient under discussion, who never awoke in the morning with his symptoms, who was seldom hungry (only 14 per cent. of the 258 patients in the series of Crain and Thorn listed hunger, supposed to be very common in this disease, as one of their symptoms) and who had characteristic attacks of weakness, giddiness and sweating on his way home from work.

It was on his way home from work that the patient did more physical exercise than at any other time of the day and it was exercise associated with fasting which allowed the symptom pattern to be repeated while in hospital. On each occasion he was relieved by rest rather than by food.

There was nothing in the history or physical findings to suggest the association of peptic ulceration with the islet cell adenoma in this patient (Zollinger and Ellison, 1955).

Diagnosis

Whipple's diagnostic triad of neurological symptoms with or without gastrointestinal symptoms, relieved by taking sugar (provided that the hypoglycaemia is not prolonged) in the presence of a blood sugar

level of less than 50 mgm. per 100 ml. must still be satisfied before the diagnosis of islet cell tumour of the pancreas can be made with certainty and the patient be submitted to operation.

Glucose tolerance curves would seem to be of little diagnostic value, as there is no agreement on what constitutes a characteristic response in a patient with an islet cell adenoma. The six hour glucose tolerance test done on 31st July would be regarded as diagnostic, according to the criteria laid down by Hoffman (1954) but would not satisfy other observers. Aird (1957) relies on the insulin tolerance test as an aid to diagnosis, but others are equally suspicious of the interpretation of this test.

It has been demonstrated that it is not the levels to which the blood sugar falls but the rate at which it falls to these levels that produces the characteristic symptomatology (Meyer, 1941). It is thus interesting to note that in our patient, following an over-night fast and a glucose load, symptoms were able to be induced when the blood sugar level was apparently above 60 mgm. per 100 ml. In the light of Whipple's triad and Meyer's work, it is also of interest that Webster and Blades (1952) have reported the case of a woman with an islet cell tumour who was comatose when the blood sugar was 64 mgm. per 100 ml.

Treatment

Although some success has been attributed to the conservative management of these patients with such measures as dietary adjustment, alloxan and, more recently, corticosteroids (Brown *et alii*, 1952), the concensus of opinion is that surgical removal of the tumour is necessary before the frequent hypoglycaemic attacks leave some irreparable cerebral damage. As a general principle, it is said that tumours of the body should be excised together with that portion of the pancreas distal to the tumour, while those adenomata in the head should be shelled out in order to prevent the formation of a pancreatic fistula. Opinion is divided as to whether radical or partial pancreatectomy should be performed in a patient who has a diagnostic history of islet cell tumour but in whom no tumour can be found at operation.

Whipple and Frantz assessed the mortality in the series which they reported in 1935 as 16 per cent. and this had fallen to 10 per cent. when Crain and Thorn wrote in 1949, describing all patients in the literature, which means that the mortality between 1935 and 1949 was considerably less than 10 per cent. and is probably not more than 3 to 4 per cent. at the present time.

SUMMARY

1. A case of an islet cell tumour of the pancreas which was successfully removed at operation is reported.
2. A brief survey of the history of this disease is given.
3. The case described emphasizes the observation that once a symptom pattern is developed in a patient with an islet cell tumour of the pancreas, it usually remains constant in that patient, even though it may not resemble the classical description.
4. The relevance of certain diagnostic tests is discussed and the importance of Whipple's diagnostic triad is stressed.

ACKNOWLEDGMENT

I wish to acknowledge with gratitude the help and encouragement of Professor C. R. B. Blackburn in the preparation of this case report and also to thank Sir Charles Bickerton Blackburn, who made the diagnosis initially and referred the patient to the Unit.

REFERENCES

AIRD, I. (1957), "Companion in Surgical Studies," 2nd Edition. Edinburgh, Livingstone.

BANTING, F. G. and BEST, C. H. (1922), *J. Lab. clin. Med.*, vol. 7, page 251.

BOYD, W. (1958), "Pathology for the Physician," 6th Edition. Philadelphia, Lea and Febiger.

BROWN, G. (1956), *Aust. N.Z.J. Surg.*, vol. 25, page 198.

BROWN, H. HARGREAVES, H. P. and TYLER, F. H. (1952), *Arch. intern. Med.*, vol. 89, page 951.

CAMPBELL, W. R. and FLETCHER, A. A. (1923), *J. Amer. Med. Ass.*, vol. 80, page 1641.

CRAIN, E. L. and THORN, G. W. (1949), *Medicine (Baltimore)*, vol. 28, page 427.

GREENAWAY, T. M., MADDOX, J. K., EDYE, B. T., DAY, E. M. and ROBERTSON, J. M. (1946), *Med. J. Aust.*, vol. 1, page 452.

HARRIS, S. (1924), *J. Amer. Med. Ass.*, vol. 83, page 729.

HOFFMAN, W. S. (1954), "The Biochemistry of Clinical Medicine.' Chicago, Year Book Publishing Company.

HOWARD, J. M., MOSS, N. H., RHOADS, J. E. (1950), *Surg. Gynec. Obstet.*, vol. 90, page 417.

HOWLAND, G., CAMPBELL, W. R., MALTBY, E. J., ROBINSON, W. L. (1929), *J. Amer. Med. Ass.*, vol. 93, page 674.

LANGERHANS, P. (1869), "Beitrage zur mikroshoposchen anatomie der Bauschspeicheldrusse." Berlin, Lange.

MAXWELL, W. (1949), *Aust. N.Z.J. Surg.*, vol. 19, page 132.

MEYER, K. A., AMTMAN, L., PERLMAN, L. (1941), *J. Amer. Med Ass.*, vol. 117, page 16.

NICHOLLS, A. G. (1902), *J. Med. Res.*, vol. 8, page 385.

RENOLD, A. E. and THORN, G. W. (1958), in Harrison's "Principles of Internal Medicine," 3rd Edition. New York, McGraw-Hill Book Co., Inc.

WEBSTER, D. D. and BLADES, A. N. (1952), *Brit. Med. J.*, vol. 1, page 307.

WHIPPLE, A. O. (1938), *J. int. Chir.*, vol. 3, page 237.

—— and FRANTZ, V. K. (1935), *Ann. Surg.*, vol. 101, page 1299.

WILDER, R. M., ALLAN, F. N., POWER, M. H., ROBERTSON, H. E. (1927), *J. Amer. Med. Ass.*, vol. 89, page 348.

ZOLLINGER, R. M. and ELLISON, E. H. (1955), *Ann. Surg.*, vol. 142, page 709.

MEGACOLON IN THE ADULT*

A REPORT OF TWO UNUSUAL CASES

By T. F. Rose

Royal North Shore Hospital, Sydney

CASES of acquired megacolon in the Western World usually involve the sigmoid colon, often presenting as a volvulus. It is a common condition in psychotics thought to be due to long standing constipation (Ehrentheil and Wells, 1955; Johnston and Gibson, 1960).

Lewitan, Nathanson and Slade (1951) say there is a high incidence of megacolon in neurological disorders such as Parkinson's disease and postulate a neurogenic factor in this disease.

Here are reported two cases of acquired megacolon in adults who were not psychotics and whose illness was not preceded by constipation. In Case 1 the left half of the transverse colon and the proximal portion of the descending colon were involved. In Case 2 the whole of the colon from the ileo-caecal junction to the recto-sigmoid was involved in a patient whose unrotated bowel still had its primitive dorsal mesentery, allowing a chronic partial volvulus to occur.

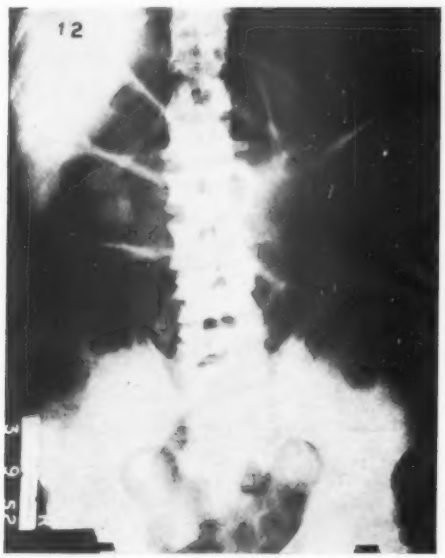


FIG. I. Case I. Plain radiograph of the abdomen showing dilated large bowel loop.

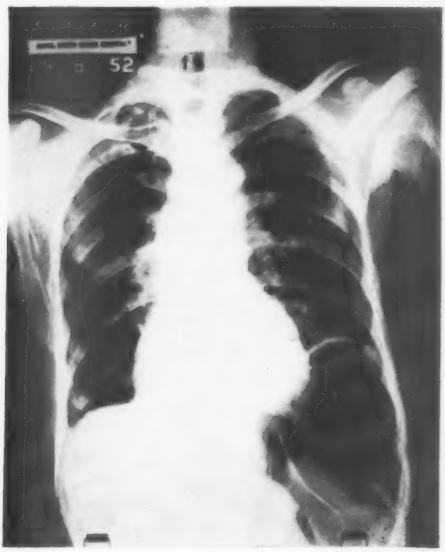


FIG. II. Case 1. Plain radiograph of the abdomen showing dilated splenic flexure of the colon.

Case 1

This sixty-eight-year-old mentally sound man was well until two years previously, since when he had suffered from recurrent attacks of precipitancy of defaecation with large fluid motions preceded by generalized colicky abdominal pains and abdominal distension particularly on the left side of the abdomen. There had never been any constipation.

Examination during such an attack of pain revealed enormous abdominal distension most marked on the left side.

A rectal examination revealed no abnormal findings. The stool was of liquid but otherwise normal faeces, there being no blood or mucus present.

*Received for publication 20th June, 1960.

A barium enema revealed a large megacolon localized to the upper descending and left side of the transverse colon (Figs. I to IV) commencing and ending abruptly.

Saline enemata relieved the condition temporarily only with the passage of fluid faeces and flatus.

After adequate preparation with thalazole and enemata, laparotomy was performed. The small bowel was normal as was the colon to the middle of the transverse colon where it became abruptly dilated to a point half-way down the descending colon where the dilated area just as suddenly ended (Fig. V).

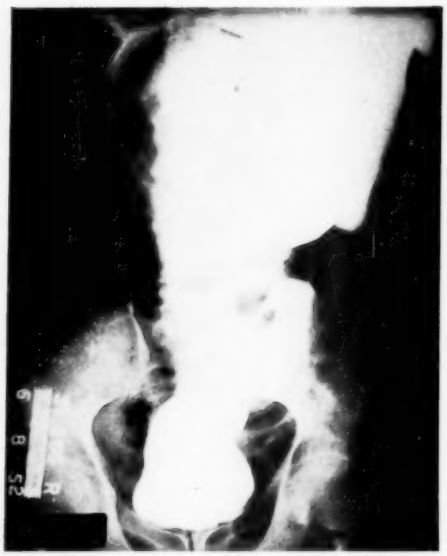


FIG. III. Case 1. Barium enema showing dilated large bowel loop on anterior view.

FIG. IV. Case 1. Barium enema showing dilated large bowel loop on lateral view.

The affected bowel was excised by cutting through normal bowel above and below, an end-to-end anastomosis being performed.

Convalescence was uneventful and normal bowel actions were obtained.

A four-year follow-up revealed that all was well, but the patient was then lost to sight.

The specimen measured 35 cm. long and was enormously dilated, its circumference being 23 cm.

Histological sections from each end of the dilated segment and the adjoining normal bowel revealed normal ganglion cells with no evidence of degeneration or fibrosis.

Comment

This patient's megacolon was localized to an unusual area and it is of interest to note that ganglion cells were not only present in normal numbers but were also normal in appearance, so contrasting with their destruction and degeneration in those cases of acquired megacolon of doubtful aetiology, common in South America where they are seen more frequently even than colonic cancer (Raia, 1955; Fisher and Swenson, 1960).

Case 2

This sixty-three-year-old female patient four years previously had had an ovarian cyst and her appendix removed. Her doctor noted at that time that the small bowel was on the right side of the abdomen and the large on the left with her caecum in the pelvis. There was no fixation or rotation of the colon and all bowel was suspended from a midline mesentery.

He especially noted at that time that the colon was otherwise normal.

The patient was then well, her motions being always normal and never constipated.

Nine days prior to admission to hospital for her present illness, she commenced to suffer from recurrent attacks of lower abdominal colic accompanied by constipation and increasing distension.

These attacks were relieved by the spontaneous evacuation of fluid motions and flatus but the attacks kept recurring with increasingly severe pain and distension.

Examination on admission disclosed marked generalized abdominal distension. Pelvic and rectal examinations disclosed a cystic mass in the pelvis, later shown to be the caecum.

The patient was vomiting and dehydrated. Radiological facilities were unavailable at that time for further investigation.

After gastric suction, intravenous fluid and electrotype replacement, laparotomy disclosed the whole of the colon from ilium to rectosigmoid to be in a state of partial volvulus with great distension.

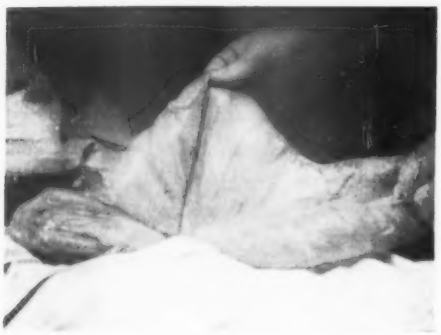


FIG. V. Case 1. Dilated loop of colon shown at operation prior to resection.

The volvulus was untwisted and seen to be viable. The embryological state of the unrotated bowel and its mesentery was confirmed.

The terminal ileum was divided, the distal end closed and the proximal end anastomosed to the rectum by end-to-side anastomosis. Fourteen days later the whole colon was excised.

The resected colon was seen to be grossly dilated and full of liquid faeces.

Convalescence was reasonable except for troublesome diarrhoea which persisted for some weeks. A two-year follow-up recently revealed the patient to be very well, passing about 4 semi-solid motions per day.

Unfortunately, the specimen was destroyed mistakenly before histological investigation of the bowel could be performed.

Comment

It is unlikely that there was any relationship between the non-rotation of this patient's bowel with retention of the dorsal mesentery and the megacolon as the latter did not occur until the age of sixty-three years, prior to which there had never been any symptoms referable to bowel dysfunction. In fact, four years previously at operation for another reason entirely, it had been especially remembered that there was no abnormality of the colon apart from its non-rotation. This prolonged period of observation before the lesion appeared was also present in Burrell's cases (1957).

It was unfortunate that the state of the ganglion cells was unable to be elicited.

SUMMARY

Two patients with acquired megacolon of unusual type are documented, one involving the left side of the transverse colon and portion of the descending colon, the other the whole of an unrotated colon.

In neither instance was there any evidence of psychosis or any history of prior constipation.

In the one investigated histologically the ganglion cells of the colon were normal.

In both instances, resection of the involved colon was performed successfully.

REFERENCES

BURRELL, Z. L. (1957), *Gastroenterology*, vol. 33, page 625.

EHRENTHEIL, O. F., and WELLS, E. P. (1955), *Gastroenterology*, vol. 29, page 285.

FISHER, J. H., and SWENSON, O. (1960), *Amer. J. Surg.*, vol. 99, page 134.

JOHNSTON, I. D., and GIBSON, J. B. (1960), *Brit. J. Surg.*, vol. 47, page 394.

LEWITAN, A., NATHANSON, L., and SLADE, W. R. (1951), *Gastroenterology*, vol. 17, page 367.

RAIA, A. (1955), *Surg. Gynec. Obstet.*, vol. 101, page 69.

ENDOTRACHEAL ANAESTHESIA FOR ADENOTONSILLECTOMY IN CHILDREN*

By B. W. GUNNER

St. Vincent's Hospital, Sydney

AS recently as 1935 Shaheen and Sydenham expressed the opinion that no anaesthesia whatsoever was necessary for tonsillectomy in children. Even if there is agreement as to the desirability of general anaesthesia for tonsillectomy, not all anaesthetists would agree that endotracheal intubation is indicated. Magill (1948) wrote "a capable anaesthetist should be able to provide adequate facilities for a capable surgeon for this operation, and protection for the patient, without intubation." It is readily admitted that many experienced surgeon-anaesthetist teams have performed thousands of adenotonsillectomies safely without intubation, and obviously the members of such teams see no reason to change to endotracheal methods.

However, endotracheal anaesthesia has advantages over pharyngeal insufflation techniques. These advantages are especially appreciated by the less experienced surgeon or anaesthetist, although it is stressed that an endotracheal tube is offered as an adjunct to, and not a substitute for, good surgery and competent anaesthesia. The advantages offered by endotracheal anaesthesia do, to a large extent, offer a solution to the three chief problems of anaesthesia for endoral surgery.

These problems are safeguarding the airway, controlling the depth of anaesthesia and preventing aspiration of blood.

Safeguarding the airway

The inadequacy of the airway in insufflation techniques is illustrated by the findings of Collins and Granatelli (1956). They reported oximetric readings which gave arterial oxygen saturation levels down to 75 per cent. using an insufflation technique, whereas no children who were intubated showed a saturation level below 90 per cent.

*Received for publication 16th September, 1960.

Prevention of aspiration

The insufflation technique necessitates hyperextension of the neck to ensure a patent airway and to control aspiration of blood. The uncertain protection afforded by this manoeuvre is illustrated by the findings of Steele and Anderson (1950). They aspirated

FIG. I. Oblique view of modified gag and Magill endotracheal tube.

the trachea in 129 children following adenotonsillectomy using an insufflation technique and recovered bloody secretions from 125 children. In 26 patients this measured 5-10 millilitres, and in 8 patients more than 10 millilitres. Similarly Collins and Granatelli were able to aspirate blood from 100 per cent.

of children anaesthetized for adenotonsillectomy by an insufflation technique, and from only 5 per cent. of those who were intubated.

Intubation affords further protection against inhalation of blood if extubation is deferred till post-operatively, the patient has been placed in the semi-prone position, and the normal protective reflexes are returning. Should inhalation of blood be suspected the trachea and main bronchi can be conveniently cleared by aspiration via the endotracheal tube prior to extubation.

Inhalation of small amounts of blood or tonsillar debris may well account for small areas of atelectasis which have been demonstrated post-operatively. This may explain some cases of post-operative fever and protracted convalescence.

An interesting, but fortunately rare complication of the hyperextended position is protrusion of an intervertebral disc (Symonds, 1953).

Control of depth of anaesthesia

The insufflation technique demands that the patient shall be well saturated with ether, prior to insertion of the gag. Otherwise anaesthesia may lighten embarrassingly if there is any difficulty inserting the gag. An even plane of anaesthesia can be maintained only if the patient is deeply anaesthetized initially for, deepening anaesthesia with the gag in place, is no easy matter. This saturation of the patient with ether implies a degree of intoxication beyond that employed in any other branch of surgery.

If thiopentone and a short-acting relaxant are used, intubation does not prolong the induction time, it affords ready control over the depth of anaesthesia during maintenance, and the means of giving oxygen or assisting the respirations in an emergency. Post-operative recovery is rapid, and the surgeon is spared the discomfort of inhaling a proportion of the ether vapour.

Disadvantages of endotracheal anaesthesia

The disadvantages of endotracheal anaesthesia are chiefly those of trauma during intubation, and the delayed complications of granulomata and sub-glottic oedema. Trauma is almost completely avoided by adequate training, avoidance of the nasal route and by the use of short acting muscle relaxants. Granulomata are very rare in children. Sub-glottic oedema is the disadvantage of endotracheal anaesthesia in children but is becoming less of a problem with care in avoiding mechanical and chemical trauma to the larynx and bacterial contamination of the apparatus used. Digby Leigh was able to report 3,900 children and infants intubated in one year with no post-operative tracheotomies and only one severe sub-glottic oedema, whilst Wrigley reviewed 12,000 intubations with no permanent sequelae.

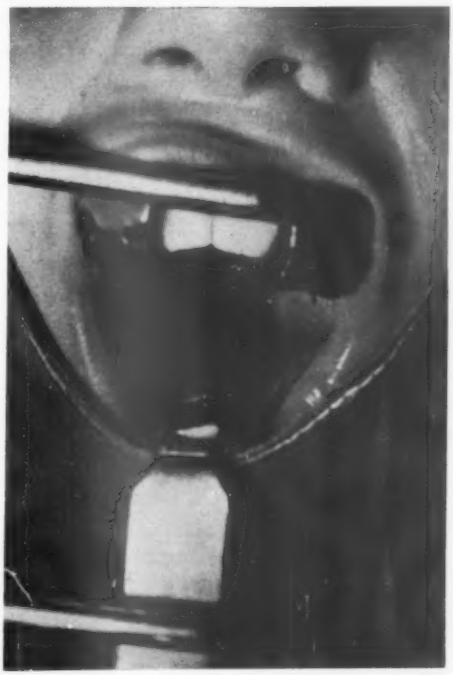


FIG. II. Endoral view of gag and tube *in situ*.

Transient hoarseness and stridor following anaesthesia for tonsillectomy are no less common after endopharyngeal insufflation than after endotracheal techniques. Presumably in the former cases the larynx is irritated by the blast of cold ether vapour and by aspirations of blood and mucus. Collins and

Granatelli recorded an incidence of 10 per cent. hoarseness and stridor after insufflation and 4 per cent. after endotracheal anaesthesia.

Techniques of endotracheal anaesthesia

Intubation may be oral or nasal. Although of some merit in adult tonsillectomy, nasal intubation has disadvantages in pediatric practice. These are trauma to narrow nasal air passages, trauma to the adenoids, possible obstruction of the tube by septal spurs, necessity to remove the tube to permit adenoidectomy, and the use of a narrower tube than in oral intubation.

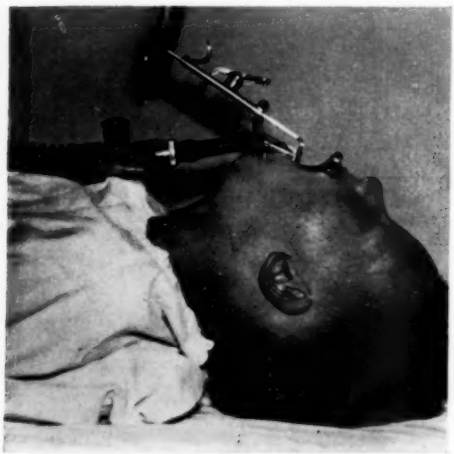


FIG. III. Lateral view of patient with gag and tube *in situ*.

A disadvantage of oral intubation is the encroachment by the endotracheal tube on to the operative field. This disadvantage can be overcome by displacing the tube and tongue towards the opposite side to the dissection (McAlpine and Bowering, 1958). The inconvenience of shifting the tube and its attachments from side to side has prevented ready acceptance of this technique.

Numerous modifications of the Davis type gag (Hawkesley and Humbey, 1943; Monro, 1948; Barton, 1955; Doughty, 1957) have been designed to restrict the trespass of the endotracheal tube on to the surgical field and to protect the tube from compression or kinking. Some surgical teams (Rotter and Mountford, 1958) simply placed the tongue blade of a Davis gag over the endotracheal tube. However, using this simple technique the tongue is difficult to orientate in the midline and the tube is easily kinked and tends to protrude from below the tongue plate.

The efficiency with which the Dott gag, designed for cleft palate surgery, accommodates the endotracheal tube is well recognized and Doughty's modified tongue piece (Doughty, 1957) is an extension of the same principle. In the majority of cases Doughty's arrangement of tube and tongue pieces works very well and is very similar to the modified Davis gag in use at the Adelaide Children's Hospital. In this modification the tongue blade contains a bevelled slot to locate the endotracheal tube. Proximally the slot is shaped to receive a special tube connection which fixes the endotracheal tube and facilitates manipulation of the gag and tube by the surgeon. A distal loop prevents herniation — and so obstruction of the tube. The endotracheal tube connection allows the anaesthetic tubing and valve to lie comfortably on the patient's chin and chest.

Magill, Oxford and armoured endotracheal tubes have all been used satisfactorily with this arrangement. The Oxford tube has some advantages in that the moulded curve prevents kinking; the oral portion, because it is thicker walled than the laryngeal, resists compression and the calculated length of the distal limb of the tube makes endobronchial intubation unlikely.

The incidence of post-intubation complications bears a relationship to the length of surgery (Pender, 1954), being less frequent with shorter than longer operations. This is no doubt a factor influencing the low incidence of complications using this technique. Follow up of the first 300 cases in the present series showed an approximately 5 per cent. incidence of transient hoarseness and one patient developed a mild sub-glottic oedema. There was no instance of a more permanent sequela attributable to the intubation.

SUMMARY

The case for and against endotracheal intubation of children for adenotonsillectomy is presented. Techniques of intubation are

discussed. A modified Davis gag, which has proved satisfactory in over 300 cases is described.

ACKNOWLEDGEMENT

My thanks are due to Mr. P. G. Jay, senior oto-laryngologist, Adelaide Children's Hospital, for his assistance in the design and testing of the modified gag.

REFERENCES

BARTON, R. T. (1955), *Postgrad. Med.*, vol. 17, page 23.

COLLINS, V. J. and GRANATELLI, A. (1956), *J. Amer. Med. Ass.*, vol. 161, page 5.

DOUGHTY, A. (1957), *Brit. J. Anaesth.*, vol. 29, page 407.

HUMBY, G. and HAWKESLEY, M. (1943), *Brit. Med. J.*, vol. 1, page 317.

LEIGH, M. D. (1959), *J. med. Ass. Georgia*, vol. 48, page 508.

MAGILL, I. W. (1948), *Brit. Med. J.*, vol. 1, page 417.

MCALPINE, D. F. and BOWERING, M. W. (1958), *Canad. Anaesth. Soc. J.*, vol. 5, page 61.

MONRO, J. S. C. (1948), *Brit. Med. J.*, vol. 1, page 267.

PENDER, J. W. (1954), *Anaesthesiology*, vol. 15, page 495.

ROTTER, K. and MOUNTFORD, L. (1958), *Lancet*, vol. 1, page 772.

SHAHEEN, H. B. (1935), *Brit. Med. J.*, vol. 1, page 37.

STEELE, C. H. and ANDERSON, J. R. (1950), *Arch. Otolaryng. (Chicago)*, vol. 51, page 699.

SYDENHAM, F. W. (1935), *Brit Med. J.*, vol. 1, page 179.

SYMONDS, C. (1953), *Lancet*, vol. 1, page 451.

WRIGLEY, F. R. H. (1958), *Canad. med. Ass. J.*, vol. 79, page 459.

HAEMOBILIA AS A CAUSE OF GASTRO-INTESTINAL HAEMORRHAGE*

By T. F. Rose

Royal North Shore Hospital, Sydney

HAEMORRHAGE from the biliary system into the alimentary canal receives but scant mention in discussions on the causes of haematemesis and melaena though it was first specifically mentioned by Nauyn as long ago as 1892. Nevertheless, this cause of gastro-intestinal haemorrhage is not uncommon and must be considered in cases of uncertain origin.

Such haemorrhage may be massive, even lethal, or it may be minimal being found as occult blood only and the cause of an obscure anaemia.

In order for haemobilia to be a cause of gastro-intestinal haemorrhage, there must be a lesion either in the liver, gall-bladder, the bile ducts or the pancreas.

There must also be a channel for the blood to reach the bowel either by the common bile duct or by a fistula from gall-bladder to duodenum. To be scientifically accurate, therefore, one should see at operation the causative lesion actually bleeding and the blood in the conducting channel. This would obviate such a mistake as occurred in the patient of Browning, Clauss and MacFee (1959) who had an aneurysm of the hepatic artery, a well-known cause of haemobilia, but whose haemorrhage per rectum was actually due to a localized area of ulcerative colitis.

This unequivocal proof of haemobilia as a cause of gastro-intestinal haemorrhage is often missing. Take, for example, the patient who has a history of melaena and in whom a probable causative lesion is found in the liver or biliary tree but in whom no blood is visible in the conducting channel and whose alimentary canal is normal. Here it is assumed the lesion is the cause of haemorrhage and this assumption is strengthened by the therapeutic trial of excision of the lesion with a follow-up revealing no recurrence of the bleeding as in some of the cases here presented (Case IV is an example of probable diagnostic error revealed in the follow-up).

*Received for publication 26th August, 1960.

Causes of Haemobilia

Causes in the liver

Numerous causes of haemobilia occurring in the liver are discussed in the literature such as primary liver cancer (Fisher and Creed, 1956) or aneurysm of the hepatic artery either communicating directly with the duct system (Kerr, Mensh and Gould, 1950) or causing a central apoplexy (Mackay and Page, 1959).

Goulston (1956) reported a most interesting patient, whom I was privileged to observe, suffering from recurrent severe and eventually fatal gastro-intestinal haemorrhage the cause of which was found at autopsy to be a large central liver hydatid cyst containing clotted blood and fibrin and whose lumen communicated with several bile ducts. The bile ducts were dilated with blood, bile and pus. Considering the frequency with which liver hydatids rupture into the bile ducts this is indeed a rare manifestation of this disease.

Trauma is an important cause of haemobilia as illustrated by the following example.

Case 1. Liver trauma causing haemobilia

This soldier suffered a perforating shrapnel wound of the right upper abdominal quadrant. Severely shocked, he was vomiting blood.

At operation, the liver was found to be literally shattered with one large piece lying free in the abdominal cavity. Both the gall-bladder, the common bile duct and the stomach contained blood but were not actually injured.

All that one could do was to place a tube down to the liver.

A stormy convalescence followed with pneumonia, wound infection and a prolonged biliary fistula with ultimate recovery.

This is not the history characteristic of "traumatic haemobilia", the term introduced by Sandblom (1948). It is usually that of a delayed gastro-intestinal haemorrhage some days or even weeks after closed injury to the abdomen, first described in detail by Sandblom (1948) and later still by Sparkman (1953), Barling (1956) and Sworn (1959).

In these cases, as a result of a deep or even subcapsular laceration, a cavity arises in the liver communicating with the bile ducts. It may also be accompanied by injury to the hepatic vascular system such as a traumatic aneurysm of the hepatic artery.

Haemangioma of the liver is another cause of haemobilia (Kerr, Mensh and Gould, 1950).

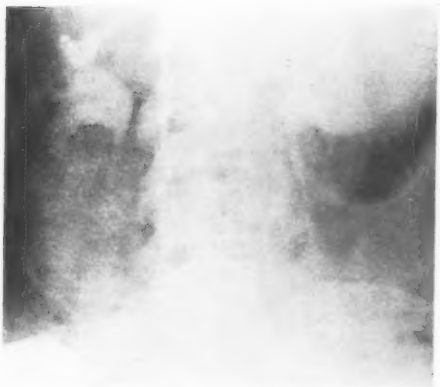


FIG. I. Intravenous pyelogram of Case 2 showing the speckled haemangioma.

Case 2. Liver haemangioma causing haemobilia

This fifty-year-old female was known to have had an abdominal mass for four years which caused intermittent aching pains in the region of the right hypochondrium.

Examination disclosed a large, smooth, rounded firm mass projecting below the right costal margin.

A plain radiogram of the abdomen revealed a large rounded soft tissue shadow in the right upper abdominal quadrant calcified in a speckled manner. The shadow moved on respiration. A barium meal revealed a normal stomach and duodenum. A Graham's test showed a well functioning, normally shaped gall-bladder behind and to the left of the mass. An intravenous pyelogram was normal, the right kidney being behind the mass (Fig. I).

Operation was advised but refused by the patient until she had a sudden melaena causing faintness and weakness following an exacerbation of the abdominal pain.

At operation a week later, with a blood transfusion, a venous haemagioma measuring three inches by two inches was excised with little difficulty from the anterior margin of the liver on a plane lateral to the gall-bladder. The mass was spongy and felt gritty due to little specks of calcification. There were no large feeding vessels and there was no obvious communication with the biliary tree.

No blood was seen in the gall-bladder or common bile duct. The alimentary canal appeared normal.

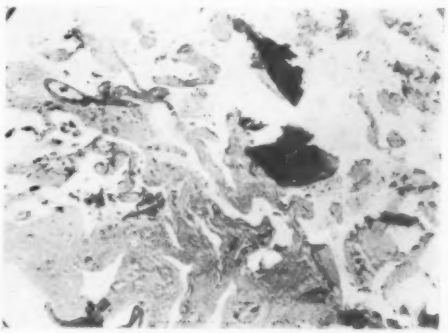


FIG. II. Photomicrograph (low power) of the haemangioma in Case 2 showing areas of calcification.

The abdomen was closed with drainage of the area. Convalescence was uneventful save for a little drainage of bile for three days.

The histological picture was that of a simple angioma with numerous areas of calcification (Fig. II).

A follow-up over eighteen months revealed no further episodes of gastro-intestinal haemorrhage.

It is surmised that some part of the angioma must have communicated with the intrahepatic biliary tree so causing a haemobilia leading to the melaena.

Causes in the gall-bladder

Haemorrhage into the gall-bladder was first reported by Nauyn (1892) from a ruptured arterial aneurysm. It has also been reported from a football kick on the abdomen (Meyer-May and Joyeux, 1939). Budinger (1925) first described massive gastro-intestinal haemorrhage due to gall-bladder

disease. Hudson and Johnson (1946) state that haemorrhage into the gall-bladder is of great clinical importance and may be due to gall-stones and, in fact, may be the only symptom or sign attributable to the gall-stones. Such haemorrhage may be overt and massive or occult only causing an undiagnosed anaemia. They present 4 such cases (though one had sarcoma of the gall-bladder as well). Heusser (1925), however, considered such bleeding to be due, not to the actual gall-stones, but to changes in the blood vessels of the gall-bladder wall.

Case 3. Calculi in the gall-bladder causing haemobilia

This seventy-year-old male had had frequent attacks of severe haematomesis and melaena necessitating blood transfusions over the previous two years.

Physical examination and radiological studies of the gastro-intestinal tract revealed no cause for this. Cholecystography was not performed.

At laparotomy the only intra-abdominal lesion found was chronic cholecystitis with the gall-bladder full of multiple smooth, facetted mixed calculi. The common bile duct was normal. Cholecystectomy was performed.

Examination of the gall-bladder revealed no blood in its lumen. There was no ulceration of the mucosa or other pathological condition of the gall-bladder apart from chronic cholecystitis.

The histological picture of the gall-bladder confirmed the presence of chronic inflammation. It also showed that the blood vessels were normal.

A follow-up over two and a half years revealed no further gastro-intestinal haemorrhages.

This case shows that pre-operatively a cholecystography and choledochography are a necessary investigation of gastro-intestinal haemorrhage of obscure aetiology.

The complications of gall-stones may also cause haemobilia as, for example acute haemorrhagic cholecystitis (Epstein, 1952: Stahl, 1959). Dr. Bernard Lake of Sydney Hospital (1960) has allowed me to mention a case in which the patient died from a massive haematemesis and melaena from a large inflammatory polyp in an abscess cavity round a cholecyst-duodenal fistula, the gall-bladder showing chronic inflammatory changes.

Cancer of the gall-bladder is rarely complicated by overt gastro-intestinal haemorrhage and large series of cases are documented in which no mention of this symptom is made (Fortner and Pack, 1958: Thorbjarnarson and Glenn, 1959: Strauch, 1960). However, Sainbury and Garlock (1948) mention melaena as an uncommon symptom in a review of 75 cases of cancer of the gall-bladder.

Case 4. Cancer of the gall-bladder probably causing haemobilia

This 55-year-old man had had occasional attacks of melaena for three years culminating in severe haemorrhage per rectum of bright red blood for the five days before admission.

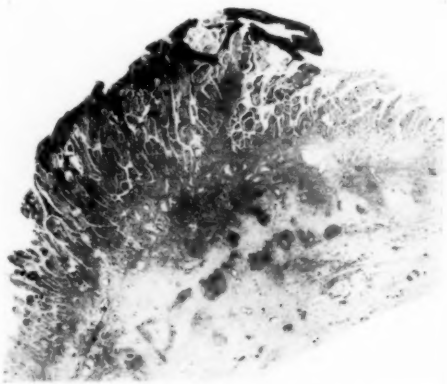


FIG. III. Low power view of the gall-bladder carcinoma of Case 4 showing haemorrhage on its surface.

Examination including sigmoidoscopy revealed only an anaemia (haemoglobin 6 gm. per cent.). Frank red blood was present, well mixed in the stool. Radiological studies of the gastro-intestinal tract were normal. Cholecystography was not performed.

At later laparotomy, the only abnormal abdominal findings were that the gall-bladder contained some blood and there was a small hard tumour palpable in its fundus though it was not visible externally. The cystic gland was not enlarged. There was no blood in the common bile duct nor in the alimentary canal which was normal.

On opening the gall-bladder, a small ulcerated tumour was found in the fundus from which bleeding was occurring into the lumen (Fig. III). Microscopical examination showed it to be an adenocarcinoma actually invading the muscle coat but not the

scrosis. Mainly well differentiated it was anaplastic in parts and was surrounded by chronic inflammatory changes (Fig. IV).

The patient was then well for ten months when he presented with a recent small, firm plaque like nodule in the skin over his sternum. Excision and histological examination revealed a picture similar to that of the gall-bladder carcinoma (Fig. V).

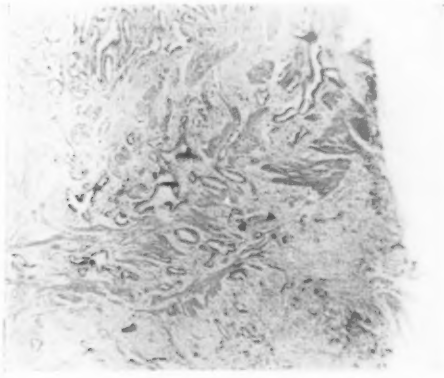


FIG. IV. High power view showing well-differentiated area of gall-bladder carcinoma of Case 4.

Three months later, just twelve months after the cholecystectomy, episodes of melaena again commenced necessitating blood transfusions. Radiological investigation of the gastro-intestinal tract and biligrafin tests were normal so laparotomy was again performed, this time to reveal no apparent abnormality at all. (There were no facilities available for cholangiography or arteriography.)

For the last eight months to date he has been well. Although the alleged haemobilia enabled an early gall-bladder carcinoma to be diagnosed, it had apparently already metastasized to the skin.

Causes in the extrahepatic biliary duct system

Post-operative haemobilia may occur especially if a T-tube is left in the common bile duct.

Case 5. Post-operative haemobilia

This patient had a cholecystectomy performed for gall-stones together with a T-tube choledochostomy after the removal of two common duct calculi.

The patient was well for seven days when severe haemorrhage occurred from the tube with a haematemesis and later melaena. As the haemorrhage did not stop, it was thought that the tube might be causing pressure in a vessel in the wall of the common bile duct. The tube was able to be removed by traction and the haemorrhage thereupon ceased.

Four similar cases of post-operative haemobilia are described by Deschamps (1959) following common bile duct exploration. In the case of Andreassen and Lindenberg (1959) bleeding did not commence until one month after operation and then required a hepatic artery ligation for its control. Other causes of common hepatic or bile duct haemorrhage causing haemobilia and gastro-intestinal haemorrhage are well documented. Barzilai and Kleckner (1956) describe a case of rupture of an aneurysm of the portal vein into the common hepatic duct in a patient with liver cirrhosis. Kehr (cited by Lichtman, 1936) reports the case of a common duct calculus eroding the hepatic artery with massive haemobilia and death. Teter's (1954) patient died from haemorrhage from a common bile duct adenoma. Huguein (1903) reported a case of cancer of the common bile duct with haemobilia.

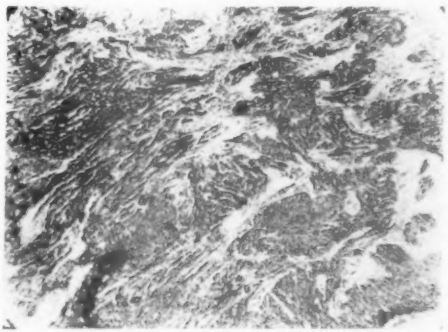


FIG. V. High power view of a field showing anaplastic area of the skin nodule in Case 4.

Stahl (1959) points out that blood in the common bile duct may spread up the bile ducts, clot and form a cast of the biliary tree and cause jaundice.

Causes in the pancreas

Cases of massive gastro-intestinal haemorrhage due to pancreatic lithiasis have been reported (Dagradi and Meister, 1959).

CONCLUSIONS

In cases of gastro-intestinal haemorrhage of obscure origin, haemobilia must be considered as a cause. As well as radiological studies of the alimentary canal, the biliary tract must be investigated pre-operatively by cholecystography and choledochography.

Whilst the possible causes of haemobilia may be obvious at laparotomy as in the cases presented here, at times they may be hidden deep within the liver.

Special investigation is then necessary on the table and may include operative cholangiography, hepatic arteriography and even portography as pointed out by Mackay and Page (1959).

SUMMARY

The causes of haemobilia presenting as gastro-intestinal tract haemorrhage are discussed. It is pointed out that the lesions may be in the liver, the gall-bladder, bile ducts and pancreas. Examples are given either from personal experience or from the literature.

Pre-operative investigation of gastro-intestinal haemorrhage of uncertain aetiology should include cholecystography and choledochography.

At operation, in obscure cases, operative cholangiography, portography and hepatic arteriography may be required.

REFERENCES

ANDREASSEN, M., and LINDENBERG, J. (1959), *Surgery*, vol. 46, page 870.

BARLING, V. (1956), *Aust. N.Z. J. Surg.*, vol. 26, page 70.

BARZILAI, R., and KLECKNER, M. S. (1956), *Arch. Surg. (Chicago)*, vol. 72, page 725.

BROWNING, L. D., CLAUSS, R. H., and MACFEE, W. F. (1959), *Ann. Surg.*, vol. 150, page 320.

BUDINGER, K. (1925), *Arch. Klin. Chir.*, vol. 135, page 782, cited by STAHL (1959).

DAGRADI, A. E., and MEISTER, L. (1959), *Gastroenterology*, vol. 36, page 269.

DESCHAMPS, J. H. (1959), *Bol. acad. argent. Cir.*, vol. 431, page 112, abstracted in *Surg. Gynec. Obstet.*, Int. Abstr. Surg. vol. 110, page 55.

EPSTEIN, H. J. (1952), *J. Amer. med. Ass.*, vol. 149, page 1132.

FIESSINGER, N., BERGERET, A., and LEVEUF, J. (1938), *Rev. Gastroent.*, vol. 5, page 383.

FISHER, E. R., and CREED, D. I. (1956), *Arch. Surg. (Chicago)*, vol. 73, page 261.

FORTNER, J., and PACK, G. (1958), *Arch. Surg. (Chicago)*, vol. 74, page 742.

GOULSTON, E. (1956), *Aust. N.Z. J. Surg.*, vol. 25, page 302.

HEUSSER, H. (1925), *Munch. Med. Wschr.*, vol. 72, page 2007.

HUDSON, P. B., and JOHNSON, P. P. (1946), *New Engl. J. Med.*, vol. 234, page 438.

HUGUEIN — (1903) cited by SCHNYDER — (1915), *Zbl. allg. Path. path. Anat.*, vol. 26, page 361; cited by HUDSON and JOHNSON (1946).

KEHR — cited by LICHTMAN (1936).

KERR, H. H., MENSH, M., and GOULD, E. A. (1950), *Ann. Surg.*, vol. 131, page 790.

LAKE, BERNARD (1960), Personal communication.

LICHTMAN, S. S. (1936), *Amer. J. digest. Dis.*, vol. 3, page 439.

MACKAY, A. G., and PAGE, H. G. (1959), *New Engl. J. Med.*, vol. 260, page 439.

MEYER-MAY, J., and JOYEUX, B. (1939), *Mém. Acad. de Chir.*, vol. 65, page 1217 (cited by HUDSON and JOHNSON (1946)).

NAUYN (1892), cited by FIESSINGER, BERGERET and LEVEUF (1938).

OWEN, H. K. (1848), *Lond. med. Gaz.*, vol. 7, page 1048.

SAINSBURY, F. P., and GARLOCK, J. H. (1948), *Surgery*, vol. 23, page 201.

SANDBLOM, P. (1948), *Surgery*, vol. 24, page 571.

SPARKMAN, R. S. (1953), *Ann. Surg.*, vol. 260, page 899.

STAHL, W. H. (1959), *New Engl. J. Med.*, vol. 260, page 471.

STRAUCH, G. O. (1960), *Surgery*, vol. 47, page 368.

SWORN, B. R. (1959), *Brit. J. Surg.*, vol. 47, page 254.

TETER, L. F. (1954), *J. Michigan Med. Soc.*, vol. 53, page 62.

THORBJARNARSON, B., and GLENN, F. (1959), *Cancer*, vol. 12, page 1009.

PULMONARY EMBOLISM IN SURGICAL PATIENTS*

By R. G. K. DE CASTRO†

Department of Surgery, Hammersmith Hospital and Postgraduate Medical School of London

DESPITE prophylactic and therapeutic measures, pulmonary embolism still occurs and most surgeons sooner or later suffer the tragic loss of a patient from this remorseless complication. Various procedures suggested to reduce its incidence include leg bandaging (Allen, 1953), or the application of elastic stockings before operation (Wilkins *et alii*, 1952; Wilkins and Stanton, 1953), prophylactic pre- and/or post-operative anti-coagulant therapy (Barker *et alii*, 1945; McCann, 1950; Short, 1952; Sevitt and Gallagher, 1959), and leg elevation to 45 degrees (Pearson, 1954).

Routine prophylaxis is justified only if it is shown that the incidence of fatal pulmonary embolism is significantly reduced by such measures and if the hazards of the measures themselves are less than the risks of embolism. Various authors have suggested that the former is the case: Wilkins and Stanton (1953) claim to have reduced the incidence of fatal pulmonary embolism by more than 50 per cent. by the use of elastic stockings, while others (Barker *et alii*, 1945; Reich *et alii*, 1945; Allen, 1953) have shown a significant decrease in thrombo-embolic complications when prophylactic anticoagulants are given. While there can be little doubt about the statistical significance of these observations, the incidence of complications of the respective prophylactic measures are not always clearly stated, although Sevitt and Gallagher (1959) report at least one major complication in 150 selected patients, and Allen describes two deaths from haemorrhage after anticoagulation therapy. Furthermore, it is not always clear (Murray and Best, 1938; Bauer, 1959; Lancet, 1959) that the major factor causing death was in fact the pulmonary embolism. Indeed, the present study suggests that such is frequently not the case.

*Received for publication, 19th August, 1960.
†Present address, Whittington Hospital, London.

Materials and methods

To investigate the incidence and role of pulmonary embolism all surgical patients admitted to Hammersmith Hospital during the five year period, 1954-1958, have been studied: they include the E.N.T., orthopaedic, urological, plastic, thoracic and neurosurgical cases, together with all the general surgical admissions. Neither obstetric nor gynaecological cases were included. The total number of surgical admissions during this period was 16,729. Autopsy was performed in 85 per cent. of the deaths. This high post-mortem rate lends particular significance to the deductions drawn from the observations, for a clinical diagnosis of pulmonary embolism is not always reliable: the high autopsy rate also keeps false positives and false negatives to a minimum.

Results

Of the 16,729 surgical admissions there were 783 deaths, and 77 of these had a pulmonary embolism. Post-mortem examination was carried out in 72 of the 77 cases: pulmonary embolism was considered to have caused death in 49, it was considered to have been contributory — but not fatal — in 21, and to have been purely incidental in 7.

Detailed examination of the case records and post-mortem reports of the 49 patients dying from pulmonary embolism showed that the cases could be divided into 3 separate groups.

Group 1 (33 cases)

In this group the life expectancy of the patients was short. Indeed, it is unlikely that any would have survived their hospital admission: many were already moribund, most had cancer usually at an advanced stage, while others had severe and progressive cardiovascular complications, a multiplicity of diseases or major post-operative complications. Many were aged or senile. Four typical case summaries are given:

Case A

Male, aged 34, admitted 17th November, 1954, with the diagnosis of polycystic kidneys, progressive uraemia, and dystrophia myotonica.

Rovsing's operation (L) under general anaesthesia performed on 18th November.

Sudden death on 19th November.

Post-mortem showed massive pulmonary embolism due to popliteal vein thrombosis and congenital cystic kidneys.

Case B

Female, aged 57, admitted on 6th December, 1954, with a stage 4 carcinoma of the left breast with deep ulceration of the chest wall.

Bilateral adrenalectomy and oophorectomy under general anaesthesia on 14th December.

Rib resection (L) for empyaema under local anaesthesia on 13th January, 1955.

Sudden collapse and death on 5th March, 1955.

Post-mortem showed massive pulmonary embolism, stage 4 breast carcinoma and a left empyaema.

Case C

Female, aged 38, admitted on 15th July, 1954, with diagnosis of:

(i) Left femoral and right popliteal arterial emboli.

(ii) Mitral stenosis, auricular fibrillation and congestive heart failure.

(iii) Severe diabetes mellitus.

The right leg pulses returned after a few hours. Left femoral embolectomy was performed under general anaesthesia on the day of admission, but the leg became gangrenous, diabetic coma supervened and the patient died five days after admission.

Post-mortem showed pulmonary embolism, left femoral vein thrombosis, mitral stenosis with acute rheumatic endocarditis and infarcts in spleen and kidneys.

Case D

Female, aged 76, admitted on 30th August, 1954, to a medical ward with hypertension (B.P. 210/110), ischaemic heart disease, and congestive cardiac failure. She was responding fairly well to treatment when she fell in the ward and sustained a fractured right femoral neck.

The fracture was pinned under a general anaesthetic on 4th October, 1954, and the patient was transferred to a surgical ward. Her convalescence was complicated by a recurrence of the congestive failure, and a wound haematoma.

Secondary suture of wound under general anaesthesia was done on 25th October.

Sudden unconsciousness on 3rd November and death on 7th November.

Post-mortem (Coroner): Extensive lung infarction with ante-mortem clots in both pulmonary arteries and thrombosis of veins in right thigh. The hip pinning was satisfactory.

Group 2 (7 cases)

In this group the life expectancy of the patients was estimated to be limited; they may have survived for a short period out of hospital for their lesions and diseases were similar to but less advanced than those in Group 1. Two representative case summaries are given:

Case E

Female, aged 61, was admitted on 1st March, 1957, with a diagnosis of carcinoma of the rectum, diabetes mellitus with peripheral neuritis and hypertension.

Hartmann's operation under general anaesthesia performed on 13th March.

On 2nd April bilateral deep calf vein thrombosis was noticed and anti-coagulant therapy was begun.

Sudden death the next day.

Post-mortem showed thrombosis of both femoral veins with massive pulmonary embolism, chronic bronchitis with cor pulmonale and a stitch abscess in abdominal wall.

Case F

Female, aged 83, was admitted on 19th June, 1954, with a pertrochanteric fracture of the right femur, and a right Colles' fracture after a fall at home.

McLaughlin pin and plate to femur, and reduction of Colles' fracture under general anaesthesia on 21st June.

The post-operative course was good but sudden death occurred on 7th July.

Post-mortem (Coroner): Pulmonary artery blocked by large ante-mortem clots and fractures of right femoral neck and right wrist.

Group 3 (9 cases)

In this group the life expectancy was considered to be good. Five representative case summaries are given:

Case G

Female, aged 49, was admitted on 31st May, 1956, to a medical ward with a diagnosis of chronic duodenal ulcer with melaena.

Transferred to surgical ward on 13th June.

Polya gastrectomy under general anaesthesia performed on 15th June.

Good post-operative course but sudden collapse and death on 19th June.

Post-mortem showed massive pulmonary embolism.

Case H

Male, aged 66, was an emergency admission on 10th January, 1958, with the symptoms and signs of four days' appendicitis and general peritonitis.

Under a general anaesthetic a gangrenous appendix was removed on the day of admission and the abdomen was drained.

The post-operative course was complicated by two days' hypotension, three days' paralytic ileus, gross wound infection and urinary retention. There was a good response to therapy but sudden death occurred on 21st January.

Post-mortem showed massive pulmonary embolism and localized areas of peritonitis following rupture of an inflamed appendix.

Case I

Male, aged 54, was admitted on 8th September, 1954, with a diagnosis of chronic duodenal ulcer.

Polya gastrectomy under general anaesthesia performed on 10th September; a friable duodenal stump was drained.

On 16th September the patient complained of left calf pain, collapsed and died a few hours later.

Post-mortem showed pulmonary embolus and a partial gastrectomy with leaking duodenal stump.

Case J

Female, aged 51, was admitted on 28th March, 1955, with a lump in the left breast. No axillary nodes palpable.

A radical mastectomy was carried out on 29th March for a spheroidal cell carcinoma with deposits in 2 of 5 axillary lymph nodes.

Sudden death on 31st March.

Post-mortem showed massive pulmonary embolism.

Case K

Male, aged 67, was admitted on 13th July, 1957, with a diagnosis of prostatic urinary obstruction.

Transvesical prostatectomy and bilateral vasectomy under a general anaesthetic was performed on 25th July with good post-operative progress.

Attack of breathlessness on 8th August. Homan's sign negative.

Sudden death on 11th August.

Post-mortem showed massive pulmonary embolism with thrombosis of deep veins of right leg.

DISCUSSION

Uncritical analysis of the case records and autopsy reports reveals the facts that pulmonary embolism developed in 77 of 16,729 surgical admissions, caused death in 49 (0.29 per cent.), and since 783 of the patients died, gave an incidence of fatal pulmonary embolism in 6.4 per cent. of deaths.

Critical analysis, however, shows that these values do not, by themselves, present a true picture, for in 33 of the 49 patients who died with a pulmonary embolism, life expectancy was otherwise very short, and in 7 others it was strictly limited. Indeed only 9 of the 49 patients could be said to have had a reasonable life expectancy.

With these qualifications therefore, it would not be unreasonable to claim that the significant incidence of fatal pulmonary embolism was really only 0.05 per cent. of the surgical admissions and 1.1 per cent. of the surgical deaths.

The higher incidences in surgical patients reported by Welch and Faxon (1941), Barker *et alii* (1945), and Kistner and Smith (1954) have not taken into account the life expectancy of the relevant patients so that the value of prophylactic or therapeutic measures may be unduly weighted. This is not meant to imply that such measures have no place in the management of surgical patients who develop venous thrombosis, but rather that the difficulties and risks of such therapy should be assessed against the lower rather than the higher incidence of pulmonary embolism deduced from this study.

SUMMARY

1. Retrospective analysis of 16,729 surgical patients over a 5-year period (1954-1958) has shown the incidence of fatal pulmonary embolism to be 0.29 per cent. of surgical admissions or 6.4 per cent. of surgical deaths.
2. Post-mortem examinations were performed in 85 per cent. of all the deaths.
3. When the records and autopsy reports of the patients who died with pulmonary embolism were considered in detail the majority of cases could reasonably have been expected to have died from the primary or associated diseases during or shortly after their stay in hospital. With this qualification the significant incidence of pulmonary embolism is reduced to 0.05 per cent. of admissions, or 1.1 per cent. of deaths.
4. It is against the smaller rather than the larger incidence that prophylactic measures should be assessed.

ACKNOWLEDGEMENTS

The patients were under the care of the consultant staff and the author acknowledges his thanks for permission to analyse the case records of the patients.

REFERENCES

ALLEN, A. W. (1953), *Surg Gynec. Obstet.*, vol. 95, page 107.

BARKER, N. W., CROMER, H. E., HURN, M. and WAUGH, J. M. (1945), *Surgery*, vol. 17, page 207.

BAUER, G. E. (1959), *Med. J. Aust.*, vol. 1, page 286.

KISTNER, R. W. and SMITH, G. V. (1954), *Surg. Gynec. Obstet.*, vol. 98, page 437.

Lancet (1959), vol. 2, page 1014.

MCCANN, J. C. (1950), *New Engl. J. Med.*, vol. 242, page 203.

MURRAY, G. D. W. and BEST, C. H. (1938), *Ann. Surg.*, vol. 108, page 163.

PEARSON, A. (1954), *Brit. med. J.*, vol. 1, page 643.

REICH, C., YAHR, M. D. and EGGERS, C. (1945), *Surgery*, vol. 18, page 238.

SEVITT, S. and GALLAGHER, N. G. (1959), *Lancet*, vol. 2, page 981.

SHORT, D. S. (1952), *Brit. med. J.*, vol. 1, page 790.

WELCH, C. E. and FAXON, H. H. (1941), *J. Amer. med. Ass.*, vol. 117, page 1502.

WILKINS, R. W., MIXTER, G., STANTON, J. R. and LITTER, J. (1952), *New Engl. J. Med.*, vol. 246, page 360.

—— and STANTON, J. R. (1953), *New Engl. J. Med.*, vol. 248, page 1087.

PERIRECTAL CELLULITIS AND RECTAL FISTULA DUE TO A SELF-ADMINISTERED ENEMA OF KEROSENE*

By E. Wilson

Sydney

Mr. E.W.S., a farmer aged 63, presented at the St. George Hospital complaining of severe pain and swelling of the peri-anal region for one week. During that time he had also experienced difficulty with micturition. A peri-anal incision had been made by his local practitioner three days previously and a few ounces of pus had been evacuated. This operation had not relieved the pain, swelling or dysuria.

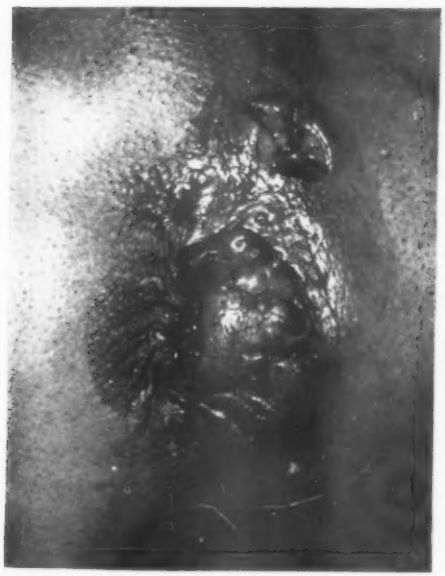


FIG. I. Photograph with the patient in the lithotomy position. Anterior to the anus a small cruciate incision had been made three days previously. The left lateral haemorrhoid is prolapsing.

On examination, his general condition was excellent yet he was obviously in great pain. The abdomen showed no abnormality; but the right buttock was very swollen, red and tender (Fig. I). There was a small cruciate incision in the peri-anal region. The left lateral haemorrhoid was prolapsed, thrombosed and infected. The prostate was hot, hard, tender and swollen and extending from its upper edge there was a tender, hard mass in which an opening about one centimetre in diameter was just palpable with the finger tip. On proctoscopy, pus was seen exuding from this opening.

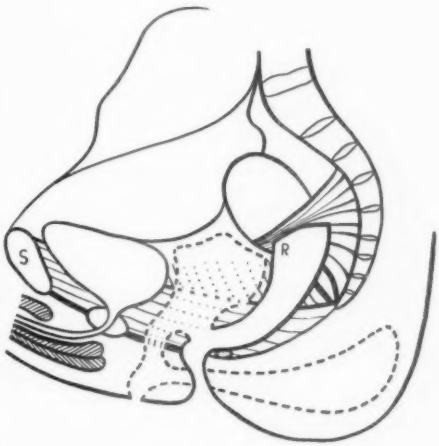


FIG. II. Diagram of the right half of the pelvis showing the extent of the inflammatory reaction (dotted line) which extended from the pelvic peritoneum to the ischiorectal fossa. The openings in the anterior wall of the rectum and in the peri-anal skin are shown. R — rectum.

Examination of the urine showed a few pus cells but no pathogenic organism was recovered on culture. The serum acid phosphatase level was 1.2 King-Armstrong units.

Under general anaesthesia the peri-anal wound was enlarged and portions of its edge and of the edge of the opening in the rectum were removed for examination.

Dr. A. Gatenby's report on the excised specimens was: "Macroscopic: some ragged fragments. Microscopic: the fragments show

*Received for publication 20th June, 1960.

chronic inflammatory granulation tissue. In one area there is a small piece of skin showing inflammatory and hypertrophic changes, but no carcinoma can be seen."

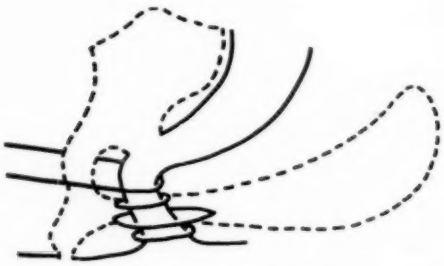


FIG. III. The inflammatory reaction (dotted line) is shown extending upwards from the opening in the anterior rectal wall to the pelvic peritoneum, downwards between the puborectalis muscles, and backwards to the right ischiorectal fossa beside the anal canal and its muscles.

DISCUSSION

When the history was first taken any local injury was denied and the provisional diagnosis at that stage was pelvic cellulitis and ischiorectal abscess possibly secondary to a malignant growth of the prostate. However, within a few days he admitted giving himself an enema of a few ounces of kerosene just after the pain in the anus had commenced. A syringe and a rigid nozzle had been used for this manoeuvre which had been followed immediately by a considerable increase in the pain.

As there was little improvement in the inflammatory condition after a fortnight, a laparotomy and a colostomy were performed. The tissues behind the bladder, seminal vesicles and prostate and below the pelvic peritoneum were all indurated but no definite abscess was detected.

During the next two months the inflammation gradually subsided and the openings in the anterior rectal wall and in the perianal region healed. The colostomy was then closed and he was discharged home free of symptoms a fortnight later. Since then the haemorrhoid, which was the start of the trouble, has been quiescent.

SUMMARY

An enema of kerosene had been self-administered in the hope of relieving the pain of a prolapsed, thrombosed haemorrhoid. Perforation of the anterior rectal wall by a hard nozzle and the injection of kerosene into the perirectal tissues resulted in a severe inflammatory reaction extending upwards to the pelvic peritoneum and downwards between the puborectalis muscles to the ischiorectal fossa. The original extent of this inflammatory reaction is shown by the broken line in Fig. II and its relationship to the muscles of the anal canal is shown in Fig. III.

ACKNOWLEDGEMENT

My sincere thanks are due to Dr. Gwenifer Bernard for her expert anaesthetics and to Mr. Roy Fluke for the drawings.

ACUTE DIVERTICULITIS WITH PERFORATION IN A JEJUNAL DIVERTICULUM*

By R. A. Rieger

University Department of Surgery, Royal Adelaide Hospital, Adelaide

THE occurrence of diverticulitis with perforation occurring in a jejunal diverticulum is comparatively rare and a case with this complication is recorded in a patient suffering from multiple jejunal diverticula.

It is difficult to assess the incidence of multiple jejunal diverticula from the literature, since there is considerable discrepancy among the published figures. Godard (1936) reports 16 cases in 13,069 autopsies. Rankin and Martin (1934), in a radiological survey, state the incidence as 1 in 25,000 X-ray examinations. Where the disease has been specifically sought after at autopsy, the incidence has dropped to 4 in 300 (Rosedale and Lawrence, 1936).

The disease is usually symptomless, about 25 per cent. of all cases having symptoms (Fraser, 1934). The commonest of these are vague abdominal discomfort, pain after meals and flatulence.

The complications of jejunal diverticula are similar to those occurring in other intestinal diverticula. They are perforation, acute diverticulitis, intestinal obstruction, volvulus of the jejunum, haemorrhage and traumatic rupture. It is also possible for a malabsorption syndrome to develop and a case of carcinoma occurring in a jejunal diverticulum has been reported (Edwards, 1939).

Acute diverticulitis is the most common complication. Milnes Walker in 1945 presented 3 such cases and reviewed 19 others from the literature.

Pathology

Non-Meckelian diverticula of the small intestine may be divided into two groups, congenital and adult. The adult forms usually occur on the mesenteric border of the jejunum and are mutiple. They are thin walled and may or may not contain muscle in the wall.

The congenital type also occurs on the mesenteric border. They are usually single and contain muscle in well-formed layers.

The mucous membrane often contains gastric or pancreatic epithelial rests (King, 1950).

FIG. I. Portion of jejunum showing three uncomplicated diverticula.

Diverticula lying on the mesenteric border may project into the mesentery or lie on one side of it. In the upper jejunum they tend to project to the right or anteriorly.

The majority arise a short distance from the mesentery and are intimately related to a vascular bundle which runs over the surface of the diverticulum.

The aetiology of jejunal diverticula is uncertain. The common association with a vascular bundle has led many writers to postulate a weakness at the point where the vessels pierce the muscle wall with herniation through the hiatus (Fraser, 1933).

*Received for publication 14th June, 1960.

The irregular association with vascular bundles is probably accidental (King, 1950), while herniation should produce a "false" diverticulum.

Many diverticula in fact contain some muscle fibres, particularly the small ones. Muscular weakness from possible vascular disease, neuromuscular incoordination or a chemical factor may be responsible (King, 1950).

The diverticula are generally wide-mouthed, so that obstruction and stasis are unlikely to occur. Acute inflammation is probably not due to obstruction or concretion within the lumen. Acute inflammatory changes in the intestinal mucosa have been observed and it is possible that a diffuse enteritis may be the initiating factor (King, 1950). These changes may be secondary or inflammation in the sac itself.

CASE HISTORY

A female, aged 57 years, was admitted to hospital with a history of acute central abdominal pain beginning 18 hours prior to admission.

The pain was sudden in onset, continuous and associated with frequent small vomits. She had undergone a cholecystectomy fifteen years before and for the last two to three years had described symptoms suggestive of recurrent cholelithiasis with vomiting in the early morning, belching after meals, but no indigestion.

On examination the patient was in considerable distress, repeatedly vomiting bile-stained fluid. The tongue was dry and furred, pulse 104/min., temperature 99°F, blood pressure 120/80 mm. of mercury.

There were abdominal signs of diffuse peritonitis with maximal guarding and tenderness just below the umbilicus. There was no particular tenderness in the right iliac fossa or per rectum. The abdomen was silent to auscultation. A plain radiograph of the abdomen showed gas in both the large bowel and terminal parts of the small bowel, with a few fluid levels. The appearances were suggestive of an ileus. A pre-operative diagnosis of ruptured Meckel's diverticulum was made.

At laparotomy through a right lower paramedian incision, an extensive purulent peritonitis was revealed. The upper jejunum contained several diverticula situated on the mesenteric border. The affected segment was 30 cms. long. There were 8 diverticula, the largest measuring 5 cm. across, flask shaped and with wide necks. The majority projected from the right side of the mesentery. The most proximal diverticulum was acutely inflamed and covered with purulent exudate. There was a small perforation in the fundus of the diverticulum.

The segment involved in the diverticulosis was excised and an end-to-end anastomosis performed. Examination of the remaining bowel failed to reveal any further diverticula.

The patient's post-operative course was uneventful.

Histology

The section showed considerable thinning of the bowel wall in the proximal diverticulum, with an acute fibrinous exudate on the serosal surface.

SUMMARY

1. A case of generalized peritonitis following perforation of an inflamed jejunal diverticulum is reported.
2. The occurrence of uncomplicated jejunal diverticulosis is not rare and symptoms associated with this disease may mimic cholelithiasis. Acute diverticulitis occurring in this situation is however an uncommon form of the acute abdomen.

ACKNOWLEDGEMENT

I wish to thank Professor R. P. Jepson for permission to publish this case.

REFERENCES

EDWARDS, H. C. (1939), "Diverticula or Diverticulitis of Small Intestine." Bristol, John Wright & Sons Ltd.

FRASER, I. (1933), *Brit. J. Surg.*, vol. 21, page 183.

GODARD, H. (1936), *Ann. Anat. path. méd.-chir.*, vol. 13, page 371.

KING, E. S. J. (1950), *Aust. N.Z.J. Surg.*, vol. 19, page 301.

RANKIN, F. W. and MARTIN, W. J., Jun. (1934), *Ann. Surg.*, vol. 100, page 1123.

ROSEDALE, R. S. and LAWRENCE, H. R. (1936), *Amer. J. Surg.*, vol. 34, page 369.

WALKER, R. M. (1945), *Brit. J. Surg.*, vol. 32, page 128.

Books Reviewed

A PRACTICE OF GENERAL ANAESTHESIA FOR NEUROSURGERY.

By ROBERT I. W. BALLANTINE, with the collaboration of IAN JACKSON. London: J. & A. Churchill Ltd., 1960. 8" x 6½", 152 pp., 68 illustrations. Price: 27s. 6d. (stg.).

The authors of this volume explain in the first paragraph of the preface, that their object is to describe in detail general anaesthetic techniques that they have found simple and efficient in neurosurgery, and to mention the work of others in this field. The techniques they describe are those currently in use at St. Bartholomew's Hospital, and there is no doubt that they fulfil this aim in an admirable manner.

The monograph commences with a brief discussion of the problems that confront the anaesthetist in neurosurgical cases. This is followed by a chapter on intracranial pressure. Here, cerebral oedema is dealt with as though it were a thoroughly understood process caused by exudation of fluid into the tissue spaces. The question mark that accompanies the words "cerebral oedema" in Figure 3 probably represents the present knowledge more accurately.

In the chapter on pre-medication and anaesthetic technique, details are carefully discussed in a very practical manner. The authors obviously have handled many cases and know the possible difficulties. Any person, taking up this branch of anaesthesia, would benefit from these details. Techniques other than their own are mentioned briefly, points for and against are given, and the reasons why they do not use them are clearly stated.

Controlled hypotension is discussed and a very reasonable attitude towards this technique is adopted. Their own method, using trimetaphan camphorsulphonate (Arfonad), is minutely described. The use of hypothermia is handled in a similar manner.

The remaining chapters deal with individual operations on the brain and spinal cord, the management of head injuries, the anaesthetic requirements of radiological investigations and the nursing of neurosurgical cases.

One criticism of the volume is the large number of pages occupied by illustrations which add little to the text. There are a number of full page reproductions of radiographs of skulls, and thirty pages are taken up with blood presure charts. In most cases, the points demonstrated are fully described elsewhere. Thus approximately one-quarter of the volume consists of non-essential illustrations.

The remaining figures are mostly line drawings and these show such ideas as postures and methods of supporting patients more clearly than would photographs.

This monograph can be recommended to all anaesthetists but particularly to those faced, for the first time, with the need to administer an anaesthetic to a neurosurgical patient, either in the X-ray department or the operating theatre.

OCULAR VERTICAL DEVIATIONS AND NYSTAGMUS.

By J. RINGLAND ANDERSON, M.C., M.D., F.R.C.S. Ed., F.R.A.C.S., D.O.M.S. Second Edition. London: British Medical Association, 1959. 10" x 7", xvi plus 189 pp., 60 illustrations. Price: 68s.

This is a second and enlarged edition of the author's "Ocular Vertical Deviations." Much new matter has been added and there is an additional chapter on congenital nystagmus, including the use of surgery for this condition.

The subjects dealt with include the types and causes of vertical deviations and disturbances of vertical movements, together with a chapter on the anatomy of the oblique muscles. Palsies of the elevator and depressor muscles are considered in detail together with their investigation and treatment.

The "A" and "V" syndromes, alternating hyperphoria and the importance of fascial sheath anomalies are discussed.

The whole book is a critical assessment of the opinions of world authorities who have written on these subjects, together with the results of the author's wide experience and deep thought on these complex matters. It should be in the library of every practising ophthalmologist.

Numerous errors in the text have been corrected in a loose page which is not included in some copies of the book.

PEPTIC ULCERATION.

By CHARLES WELLS and JAMES KYLE. Edinburgh: E. & S. Livingstone Ltd., 1960. 10" x 7", 260 pp. Price: 42s. (stg.).

The surgical treatment of peptic ulcer is a challenge to the surgeon, the physician, the pathologist and the radiologist. In skilled hands surgical treatment is usually most successful. But there are the unfortunate few, some 5-15 per cent., who are not improved or who are made worse by operation — these provide the challenge. In their excellent publication, "Peptic Ulceration," Charles Wells, James Kyle and their colleagues, present and past members of the University of Liverpool, England, have made a wise and helpful appraisal of the present status of surgery, and point the way for the future. Professor Wells is cautious regarding the choice of the operation for duodenal ulcer: "It remains for the surgeon to decide for himself, in the circumstances in which he finds himself, the operation which he will of choice routinely employ in complicated duodenal ulcer. The beginner would be well advised to adopt vagotomy and gastroenterostomy, to be unhurried, careful and thorough. Many, because of their training, and the practice of those with whom they worked, will want to follow Polya." For gastric ulcer he wisely advocates hemigastrectomy with excision of the ulcer and gastro-duodenal re-anastomosis. Professor Wells concludes his chapter by stating "the last word has not been said" and urges that new methods should be carefully assessed by controlled clinical trial.

In support of this call for improved surgery, the book naturally passes on to sinister chapters, "The early complications of operations on the stomach," "Alimentary function following gastric operations," "Delayed complications of operations on the stomach" and "Recurrent ulceration" (after operation). It is indeed a relief to reach the end of this gloomy saga to encounter a delightful appendix which includes the famous letter of 1881 from Professor Billroth to Dr. Wittelshöfer: "Dear Colleague, I willingly accede to your request for information about the resection of the stomach (for cancer) which I carried out on 29th January this year . . . " Also in this book are splendid chapters on the aetiology and pathology of peptic ulcer, and the clinical features.

This book is highly recommended to all who treat and study peptic ulceration and the authors and publishers are to be congratulated for giving us of Britain's best.

SURGICAL NURSING AND AFTER TREATMENT.

By T. EDWARD WILSON. Eleventh Edition. London: J. & A. Churchill Ltd., 1960. 8" x 5", 618 pp., 361 figures. Price: 30s. (stg.).

This standard and excellent textbook by the late Rutherford Darling has now been edited and completely brought up to date by T. Edward Wilson.

This is not a textbook of surgery but essentially a description of modern surgical nursing and surgical theatre technique with its accompanying appliances and equipment — all most necessary for the student nurse to learn.

The presentation is excellent, the large type making for easier reading and study. The drawings in black and white are clear, distinct and in most cases self-explanatory: likewise the numerous photographs and illustrations of surgical instruments.

The classifications of operations, complications, pre-operative and post-operative treatments are excellent and are a great attraction to the student nurse.

The chapters on surgical nursing, operations and surgical technique give in detail all the practical procedures which every nurse should know.

Dr. Gwenifer Bernard, an anaesthetist, has added three superb chapters to the book.

Chapter 12, devoted to the anaesthetic equipment, is perhaps unnecessary for the student nurse, but essential for the sister in charge of the theatre, and for those studying for the post-graduate theatre course. Nowhere else have I found such essential details so excellently classified.

Chapter 13, on pre-operative treatment, is really a must for every trainee, but the plan for the treatment of cardiac arrest, so prominent in the lay press recently, is really more for the surgeon than the trainee nurse.

Dr. Bernard's third chapter, likewise is a must for every student nurse. It seems that a question from this chapter appears on the final examinations every year.

The notes on the care of a colostomy and ileostomy are most helpful as many patients ask the nurse looking after them what management is necessary.

The description of splints and other orthopaedic appliances and their application is good and with the accompanying chapters on fractures and bone surgery give a balanced orthopaedic section to the book.

The specialities, eye, ear, nose and throat are not neglected and are fully described with the appropriate nursing treatment.

The article by Dr. Harold Ham on the nursing of patients undergoing radiotherapy and the helpful notes by Dr. Skipton on the collection by the nursing staff of pathological specimens make this a most complete book on surgical nursing.

The lack of coloured plates in such an excellent book is noticeable and I feel that their addition would put this book in the front rank of nursing volumes.

One can have nothing but praise for this book which every nurse and hospital library should possess.

MANUAL OF HAND INJURIES.

By H. MINOR NICHOLS. Second Edition. Chicago, U.S.A.: Year Book Publishers Inc., 1960. 9" x 6", 352 pp., 180 illustrations. Price: $9.50.

The second edition of this manual has maintained its high standard of readability and usefulness. Improvements in practical treatment appear in each chapter and in particular the treatment of crush and avulsion injuries are now treated more clearly with the inclusion of improved methods of plastic and reconstructive surgery.

Throughout this manual the author has relied entirely on his personal experience which lends authority to each method described and by its very practicality this book should retain its place as a most useful guide for surgeons concerned with the care of the injured hand.

A SYNOPSIS OF OBSTETRICS AND GYNAECOLOGY.

By ALECK W. BOURNE, M.A., M.B., B.Ch., F.R.C.S.Eng., F.R.C.O.G. Twelfth Edition. Bristol, England: John Wright & Sons Ltd., 1959. 7¾" x 4¾", 632 pp., 167 illustrations. Price: 35s. (stg.).

The law of supply and demand operates in medical books as it does in other commodities. The need of students for potted knowledge with which to brush up before the exams is a continuing one and the fact that this book is in its 12th edition indicates that it continues to satisfy this need.

It is a summary of sound British obstetrical and gynaecological practice and no one in their right senses has any major disagreement with this. But every reviewer will find some aspect of the subject where he would have hoped for a different emphasis. In this instance the complaint is that there is not sufficient instruction about, or emphasis on, the value of local anaesthesia in obstetric practice.

Extending the use of local anaesthesia can lessen maternal and foetal mortality and morbidity and had this book stressed this fact with more vigour its value would be enhanced.

Book Received

SURGERY FOR NURSES.

By JAMES MORONEY. Seventh Edition. Edinburgh and London: E. & S. Livingstone Ltd., 1961. 8½" x 6", 792 pp., 621 illustrations. Price: 32s. 6d. (stg.).

bone
the

not
opri-

g of
pful
sing
most

lent
tion
sing

book
pos-

ago,
6",

ned
Im-
ach
and
vith
and

lied
nds
ery
s a
the

Ch.,
tol,
3",

cal
eed
to
nd
di-

nd
ght
But
ect
sis,
not
lue

en
nd
its

gh
x
).